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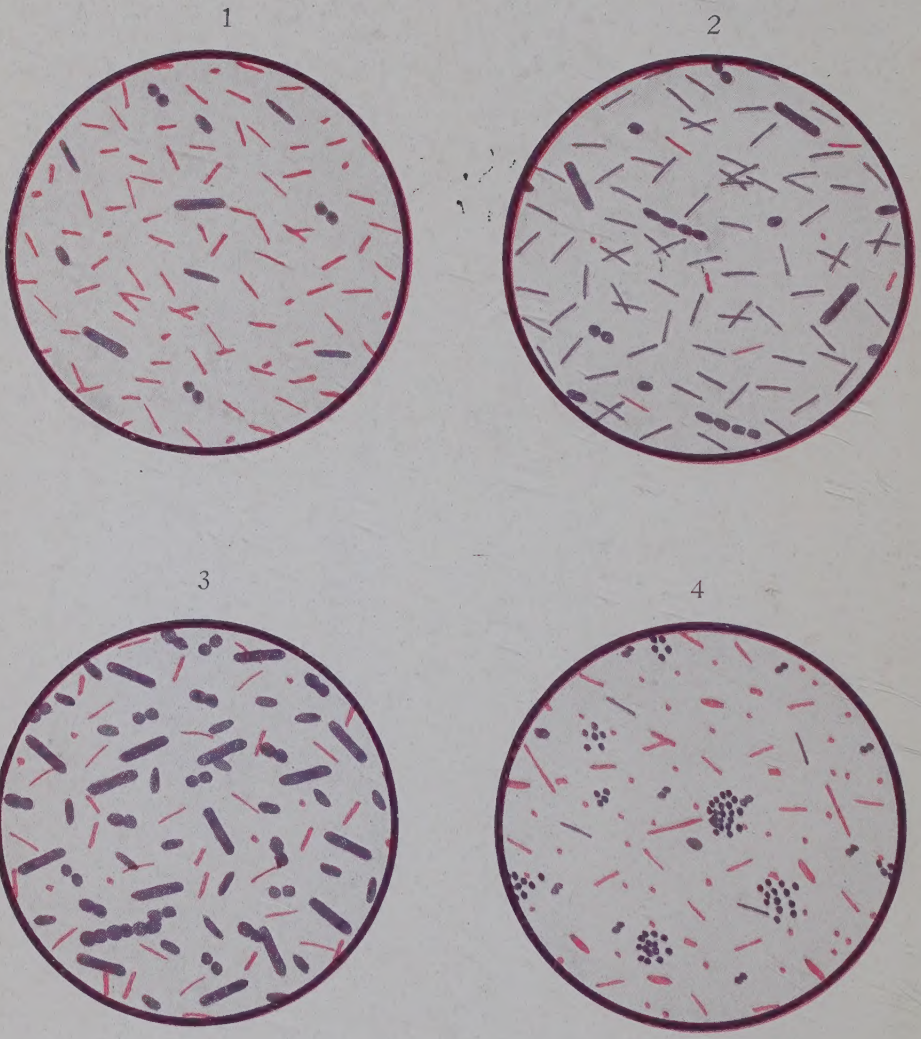

Specific bacterial infections of the human intestinal tract. Fig. 1, Bacillus coli communis, pyogenic form. Fig. 2, Bacillus putrificus. Fig. 3, Bacillus aërogenes capsulatus. Fig. 4, Streptococcus fecalis. Specimens, Gram stained.

DISEASES OF THE INTESTINES

AND

LOWER ALIMENTARY TRACT

BY

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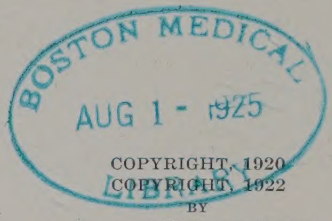

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TO THE

PRACTITIONERS OF MEDICINE

I RESPECTFULLY DEDICATE

THIS VOLUME.

OPTIMI AMICI HUMANI GENERIS

PREFACE.

THERE have been so many requests for a work on the intestines and lower alimentary tract, and my earlier book on the stomach and upper alimentary tract having met with such a gratifying reception on the part of the profession, I have felt it a duty to write this volume. In observing this obligation, a real responsibility goes with it, one I would not have met fairly by presenting simply a compilation of the various subjects. Most of the text-books on digestive disorders are to a great extent on this plan, and while perhaps they meet a desire which my work on the stomach, and now this one on the intestines, will not, nevertheless I feel that the personal equation to and the experience in the subjects outlined are, after all, the higher standards for an author to assume. There will be found in this volume the work of others which I am aware is superior to mine, which I hope to advance still further. There are presented exceptions, additions, and different points of view to many who have worked and written on the subject, and some of the matters rather generally accepted will be here presented in modified form. The list of subjects has suggested to me that considerable detachment offers the easiest plan of presentation, and in a few of them, the only one. This division is regrettable to me, because in this field of work one disease or disorder often is interwoven with another. Were the subjects few or simple, a picture of the whole might be made, but their complexity and variety is such that this is not entirely possible in a book, although it may be when teaching personally in contact with cases and ample laboratory facilities. Still, I have done my best to correlate the subjects and to so write upon them that the work will continue to serve as a guide, for it has been my intention to keep the context as clinical and close to the actual cases as possible.

ANTHONY BASSLER.

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CHAPTER I.

Topographical and Histological Anatomy.

THE SMALL INTESTINE.

The small intestine is divided into the duodenum and the jejuno-ileum. The average length in the male is 6.8 meters (22 feet 6 inches), and in the female nearly 15 centimeters (6 inches) more. This excess, however, is probably of no importance. In the male the extremes are 9.7 meters (31 feet 10 inches) and 4.7 meters (15 feet 6 inches). The outer wall of the tube is regular, without sharp folds or sacculations beyond the duodenum. The circumference is greatest in the duodenum (not always at the same point), beyond which it gradually decreases, the diameter of the gut at its lower end being nearly one-third smaller than at the beginning.

The small intestine, like other parts of the alimentary tube below the diaphragm, consists of four coats, the mucous, the submucous, the muscular and the serous.

The muscular coat, in addition to the glandular structures, possesses folds and villi that not only greatly increase its surface, but also contribute peculiarities which aid in differentiating typical portions taken from various regions. The epithelium covering the free surface consists of a single layer of cylindrical cells which exhibit a striated cuticular border next to the intestinal lumen. In many places, especially over the villi, mucus-producing goblet-cells with the ordinary epithelial elements are found. The tunica propria of the mucous coat resembles lymphoid tissue, being composed of a connective-tissue reticulum containing numerous small round cells similar to lymphocytes. This stroma fills the spaces between the glands and forms the core of the villi over which the epithelium stretches. The deepest part of the mucous coat is comprised by the muscularis mucosæ, in which an inner circular and an outer longitudinal layer are distinguishable.

The villi are minute projections of the mucous membrane, barely visible to the unaided eye, and their presence imparts the characteristic velvety appearance to the inner surface of the small intestine. They are more numerous in the duodenum and jejunum and less frequent in the ileum. They appear in the duodenum immediately

beyond the pylorus, but reach their greatest development in the second part where they measure from 0.2 to 0.5 millimeters in height and from 0.3 to 1 millimeter in breadth. In the jejunum they are conical and somewhat laterally compressed, while in the ileum their shape is cylindrical, filiform, or wedge-like. The villi are projections of the mucous coat alone, and consist of a framework of the lymphoid

Fig. 1.—Section through the small intestine of a dog. *A*, The mucous membrane. *B*, The submucosa. *C*, The circular muscle fibers. *D*, The long longitudinal muscle fibers. *E*, The peritoneum. Note the lacteal vessels in the left villus, the lymph channel in the center, and the communicating lymph vessels through the internal muscular coat.

stroma-tissue, covered by columnar epithelium, which supports the absorbent and the blood vessels, together with the involuntary muscle. Each villus is supplied by one to three small arteries, derived from the vessels of the submucosa, which break up into a capillary network lying beneath the peripheral layer of the stroma. The blood is returned usually by a single vein, which, beginning at the summit by the confluence of capillaries, traverses the central

part of the villus and becomes tributary to the larger venous stems within the submucous coat.

The absorbent, chyle-vessel, or lacteal, is the lymph-vessel occupying the center of the villus and is surrounded by muscular tissue and blood capillaries. The muscular tissue within the villus, prolonged from the muscularis mucosæ, forms a layer of slender fiber cells, longitudinally disposed, which surrounds the central chyle-vessel, and serves to empty the contents of the lymphatic vessels by contraction.

Fig. 2.—Human small intestine showing villi; base structure and portion of the submucosa. ×70.

The valvulæ conniventes, within the duodenum and the jejuno-ileum, greatly increase the secreting and absorbent surface of the mucous membrane; they also retard the passage of the intestinal contents, thereby facilitating the digestive processes. These transverse folds begin in the second part of the duodenum and consist of duplicatures which involve not only the entire thickness of the mucosa, but contain a central supporting projection of the submucous coat. The majority of the valves do not extend more than two-thirds or three-fourths of the circumference of the gut. Their ends are usually simple, but may be bifurcated. They are most numerous in the middle of the duodenum and gradually become fewer with long spaces between as descent is made into the small intestine.

In the structure of this part of the intestine are found glands, which have been divided by anatomists into true and false ones. The former are secreting glands, known as the glands of Lieberkühn and of Brunner; the latter are more or less extensive accumulations of adenoid tissue, and are appropriately spoken of as lymphatic nodules or follicles.

The glands of Lieberkühn are simple tubular depressions which are found not only throughout the entire small intestine, but in the large as well. They are closely set, narrow, and extend through the thickness of the mucous membrane as far as the muscular layer. They are lined by a single layer of columnar cells directly continuous with those covering the villi. They take no part in the absorption, never containing any fatty particles during periods in which such substances are seen within the epithelium of the villi.

The glands of Brunner are often spoken of as the duodenal glands, and are limited to the first division of the small intestine. They begin shortly beyond the pylorus, where they are most numerous and extensive, and gradually decrease in number and size, being sparingly present beyond the opening of the bile-duct and entirely wanting at the lower end of the duodenum. These glands are supposed to be direct continuations of the pyloric glands of the stomach, with which they agree in all essential details. The Brunner's glands chiefly occupy the submucosa, the migration taking place at the pyloric ring. The duodenum, therefore, possesses a double layer of true glands—those of Lieberkühn within the mucous coat, beneath which, in the submucosa, lie those of Brunner. It is more than probable that the glands of Lieberkühn are not concerned in digestion, while those of Brunner have some function, the nature of which has not been accurately found out.

Lymph-nodules are found separately or collected into considerable masses known as Peyer's patches. The solitary nodules vary greatly in number and size, sometimes being present in profusion in all parts of the small intestine, at other times, almost wanting. They are most numerous in the middle and lower parts, and appear as small whitish elevations, spherical or pyriform in shape. Villi are wanting over the prominence of the nodules. Peyer's patches are collections of solitary lymph-nodules, the individual follicles being blended by intervening adenoid tissue. They, like the solitary glands are most numerous in the lower half of the small intestine, especially near the lower end of the ileum. They are usually found in bunches of about thirty although as few as eighteen and as many as eighty-one have been counted.

The submucous coat is lax, but not enough to allow the displacement of the valvulæ conniventes, except at the lower part. It contains the blood- and lymph- vessels and the nerve-plexus of Meissner.

The muscular coat, about 0.4 millimeters in thickness, consists of an outer longitudinal and an inner circular layer. The latter is some two or three times as thick as the former, and is rather regularly arranged. The thin longitudinal layer, thickest at the free border, is often imperfect, especially at the attachment of the mesentery. The muscular layer diminishes in thickness from above downward.

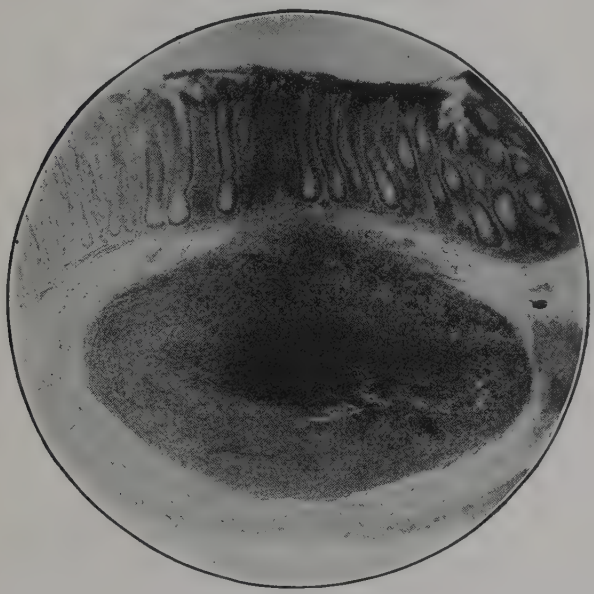

Fig. 3.—Section through a solitary gland from the small intestine of a dog. ×70.

The serous coat, with the exception of that of the duodenum, completely surrounds the gut except at the line of attachment of the mesentery, where the two layers of peritoneum diverge, leaving an uncovered space between them just large enough for the passage of the vessels and nerves.

The blood-vessels supplying the small intestine are distributed to the walls of the tube in a manner closely agreeing with the arrangement found in the stomach. The same general plan also applies to the large intestine. The arteries, after supplying the serous

coat, penetrate the muscular tunic to reach the submucosa. Within this, branches arise, which in conjunction with those directly given off during the passage through the muscular coat, supply the muscular tissue. The larger and more important arterial twigs from the vessels of the submucosa enter the mucous coat, in which they break up into capillaries forming a network surrounding the gland-tubules and supplying the muscular and stroma tissue; others pass directly toward the villi, which they enter and supply by capillary networks occupying the periphery of the projections. The veins commence within the mucosa beneath the epithelium, and gradually enlarge as they descend, and become tributary to the large veins within the submucosa. The latter follow the arteries in their passage through the muscular tunic, uniting to form the larger emergent venous channels which accompany the arterial trunks between the peritoneal folds.

The lymphatics of the small intestine, known as the lacteals, from their conspicuous milky appearance when filled with emulsified fat during certain stages of digestion, begin as the absorbent or chyle-vessels within the villi. In addition to these, radicles commence within the stroma-tissue of the mucosa, in which the lymphatics form a plexus in the plane of the muscularis mucosæ. The emergent lymphatics form larger vessels within the serous coat, which pass to the lymph-nodes situated between the peritoneal layers, and from these smaller lymphatic masses efferent vessels converge to the larger mesenteric lymph-nodes at the root of the mesentery.

The nerves supplying the small intestine, derived from the solar plexus and consisting of both medullated and non-medullated fibers from the cerebrospinal and sympathetic systems, closely follow the disposition observed in the stomach. After piercing the other longitudinal layer they form the intramuscular plexus of Auerbach, consisting of both varieties of fibers and microscopic sympathetic ganglia. The nerves then pass obliquely through the circular muscular layer and form within the submucous coat the plexus of Meissner. From the latter, non-medullated fibers enter the mucous coat and are distributed as periglandular and subepithelial networks, as well as supplying the muscular tissue, in which, according to Berkeley, additional special end-organs exist.

THE DUODENUM.

The duodenum is the beginning of the small intestine. It describes a C-shaped course, the open portion of the C pointing upward and to the left. The part beginning immediately at the

stomach is free, but a little farther back this portion of the gut is suspended from the liver by the duodeno-hepatic ligament, which is the free border of the lesser omentum, containing the portal vein, the hepatic artery, and the bile-duct with the connective tissue about them. The duodenum is therefore nearly a ring, suspended at two points; one, at the duodeno-hepatic ligament; second, at the duodeno-jejunal angle. This portion of the intestine is divided into four parts.

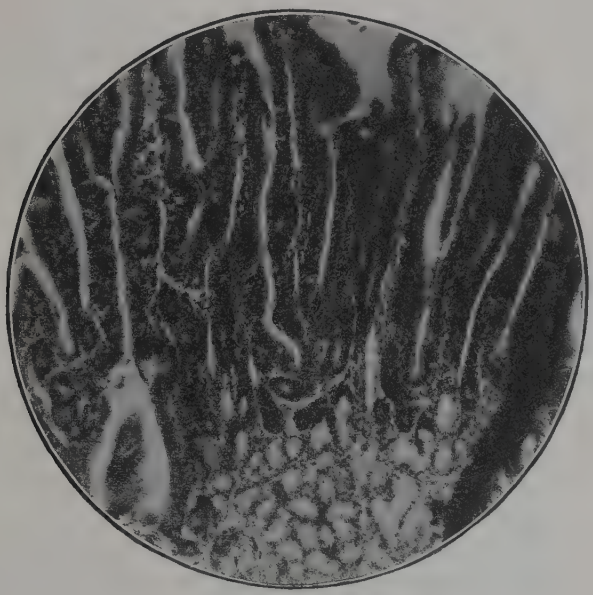

Fig. 4.—Human duodenum, showing villi.

The first part is often egg-shaped, narrowing at the ends. Its main direction is backward, slightly upward and to the right reaching the first lumbar vertebra. It is movable according as there is contents or absence of contents in the stomach. The second part descends vertically, forming an acute angle with the first. It is bent so sharply that a fold of the entire thickness often projects into the gut at this point. It lies on the right side of the vertebral bodies beside the vena cava, and borders the head of the pancreas, which it may overlap in the front. In this portion of the duodenum, the bile and pancreatic ducts open. The third part curls around the spinal column, passing forward to its front and then to the left with a slight ascent till it reaches the aorta, and helps to enclose the head of the pancreas. The fourth part begins at an obtuse angle with the third

and ascends in front of the spine to the top of the second lumbar vertebra. It overlaps the aorta and usually ends directly over it, or just at its left edge. The duodenum ends in a sharp turn, the duodeno-jejunal flexure. The very top of the gut at the bend is suspended from the left crus of the diaphragm and from the areolar tissue about the celiac axis by the duodenal suspensory muscle of Treitz, a small triangular band of muscular and fibrous tissue, which reaches the gut where it is covered by peritoneum, and is said to join the layer of longitudinal muscular fibers.

The shape of the duodenum allows food to rush through it from the stomach, as well as fluid poured into it from the liver and pancreas to accumulate, and thus acts as an S-trap to prevent the passage of gases from the intestine into the stomach. The first part of the duodenum is covered by peritoneum; the second and third parts may be said to be posterior to it, while the fourth is mostly covered. The interior of the duodenum is smooth in the first part and overlies the glands of Brunner. The villi are small at the beginning, but soon attain their complete size. In the second part, in the back or inner wall is found the bile-papilla, about 8.5 to 10 centimeters, or 3½ to 4 inches beyond the pylorus, or rather below the middle, through which the common bile-duct and the duct of the pancreas pass to open by a common orifice. It is guarded by a papilla which is almost always overhung by a valvular fold. The accessory duct of the pancreas often opens 2 or 3 centimeters above the main one through a much smaller and inconstant papilla.

The blood-vessels are derived from the celiac axis. The stomach is supplied chiefly by the gastric and splenic arteries, and the duodenum by the hepatic with the help of a recurrent branch from the superior mesenteric.

THE JEJUNO-ILEUM.

The jejuno-ileum comprises that part of the small intestine between the duodenum proper and the cecum. It is encased by the various coats of the peritoneum and attached to the posterior abdomen by means of a mesentery. The free or intestinal border of the mesentery is some 6 meters or about 20 feet long. Near its origin, in the first six inches of the intestine, the mesentery reaches a breadth of from 12 to 15 centimeters (5 to 6 inches). At the lower end its breadth is more uncertain, being usually slight, only from 2.5 to 5 centimeters for the last six inches. It increases with age, presumably concurrently with the increase of girth. The mesentery contains vessels and nerves as well as lymphatic nodes between its

folds; these structures may lie in a considerable mass of fat, adding to the thickness, which is much greater on account of the size and number of the vessels, in the upper part than in the lower.

The arteries of the jejuno-ileum are branches of the superior mesenteric, which enter the mesentery below the pancreas. The vessels for the gut are straight ones arising from the arterial arches. They run without anastomoses to the edge of the gut, where they break up into bunches of slightly diverging branches. All of these usually go to one side of the gut, each alternate vessel taking a different side, although sometimes a vessel may send branches to both sides. Anastomoses in the walls of the gut between the branches of neighboring arteries are not numerous, and occur only between very fine vessels, except opposite the mesentery, where vessels of the different sides meet. The distribution of the veins is essentially the same.

The lymphatics are large and numerous, and empty into the mesenteric nodes with which they connect. These nodes vary in number from one to two hundred, the largest lying near the root of the mesentery, from which position they grow smaller as they approach the free edge. The nerves of the entire small intestine are from the solar plexus. They receive many cerebrospinal fibers through the splanchnics.

There is a protrusion from the ileum, shaped like the finger of a glove, which is known as Meckel's diverticulum. It is the remnant of the vitelline duct, which at an early stage connects the gut with the yolk-sac. It springs most frequently from the free border of the bowel, sometimes however, from the side, and as a rule, but not invariably, is composed of all the intestinal coats. Its location is within 1 millimeter (on an average 82 centimeters) of the cecum. The diameter of the diverticulum is usually that of the gut, but it may be less and it may taper into a conical form. Its length may be as long as 17.5 centimeters (7 inches) although usually it is half that, or even less. As a rule, its end is free, but often a delicate band extends from its apex to the umbilicus or to some of the contents of the abdomen, most often to the mesentery.

THE LARGE INTESTINE.

The large intestine is distinguishable from the small, not so much by its greater size, as by being sacculated excepting perhaps at the sigmoid flexure. The length of the large intestine from the root of the appendix to the beginning of the rectum is, according to Treves,

about 1.4 meters (4 feet 8 inches) in man and somewhat less in woman. The extremes are 2 meters (6 feet 6 inches) and 1 meter (3 feet 3 inches). Excluding the dilated part of the rectum, the capacity decreases from above. The diameter, however, owing to the extreme contraction is very uncertain.

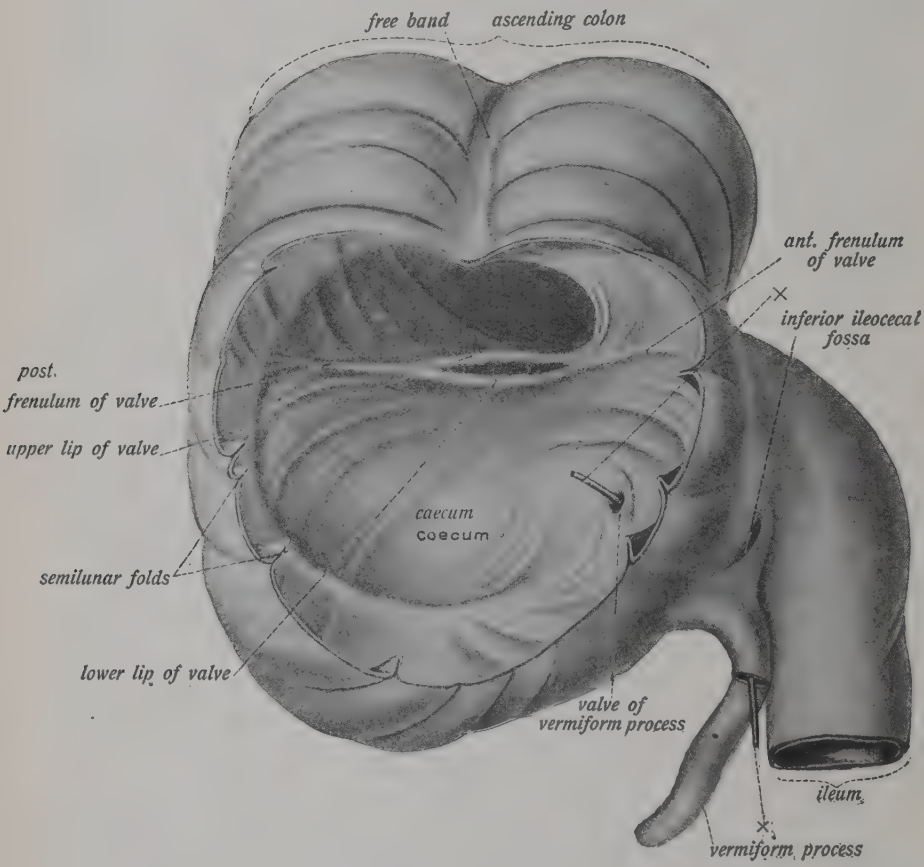

Fig. 5.—The ileocecal valve. (*Sobotta-McMurrich.*)

The structure of the mucous coat of the large intestine agrees essentially with that of the small, the chief difference being the absence of villi, in consequence of which the velvety appearance imparted by the latter is not seen in the colon. The valvulæ conniventes are also wanting, although there are projections into the large gut involving all or a part of the coats internal to the serous tunic. The glands of Lieberkühn resemble those of the small intestine, but

are larger and form a more regular and less interrupted layer of parallel tubules. The largest ones are found in the rectum where they are conspicuous about the glands of Lieberkühn. In the colon is a lining in which a large number of goblet-cells are found, which often exist in such profusion in the middle and upper part of the tubules that the ordinary cells are almost entirely replaced. The presence of a large number of goblet-cells accounts for the considerable amount of mucus normally poured into the large intestine.

The lymphatic tissue occurs in definite nodules only, Peyer's patches being absent within the large intestine.

The muscular coat consists of a thicker layer of internal circular fibers and of an external longitudinal one, the fibers of which are in most places collected into three bands. Beginning in the cecum and at the base of the vermiform appendix, the three bands, or teniæ, continue along the large intestine as far as the sigmoid flexure, over which, and the rectum, the bands become only two and are no longer sharply defined. In the rectum one is on the front, and the other, the stronger, behind. The circular fibers increase greatly toward the end of the rectum.

There is nothing special to be added regarding the serous coat, excepting that, fringing the colon there will be found bags of peritoneum containing fat, which are known as the appendices epiploicæ. They are found particularly on the inner aspects of the ascending and descending colon and on the lower one of the transverse colon.

The first part of the colon is called the cecum, or blind gut, and is the part hanging downward at the junction of the ileum and colon, from which the vermiform appendix arises. The ileum opens into the large intestine by a transverse orifice placed internally and somewhat posteriorly. From the top of the ileum a deep furrow passes posteriorly partly around the gut, and a less marked one is found in front. The average length of the cecum in the adult is between 6 and 7 centimeters (about 2½ inches) and its breadth about 8 centimeters (3⅛ inches). In the adult, the bands of the colon are continued into the cecum and terminate at the origin of the appendix. One band is in front and the other two externally and internally at the back. There are two chief forms of cecum, with several minor modifications; the first is a persistence of the fetal type, in which the cecum has the shape of a cornucopia bent to the left with the tapering end continued as the vermiform appendix. The other, which is the usual, occurring in from 91 to 94 per cent. of adults, is due to the part between the external and the anterior band growing out of all proportion, so that the pouch between them becomes the lowest part, apparently the

apex, the appendix arising from the internal posterior side near to the ileum.

The end of the ileum is thrust in between the cecum and the colon at an angle. The ileum, near to its end, lies between the surface of the cecum below and the lower swelling of the colon above; thus the upper of the two lips of the elliptical opening is composed of colon and ileum, the lower of ileum and cecum. They form prominent shelf-like projections into the large gut, opposite the external furrows, and constitute the ileocecal valve.

The orifice of the vermiform appendix is variable. In some cases the cecum narrows to it so gradually that it is hard to say where it begins; in others it begins suddenly with an oval or round opening measuring from 5 millimeters or less, to 1 centimeter or more. There is sometimes a valve at the orifice, but this is not a true valve, but a projection made by the wall at the union of the cecum and appendix in the entering angle when it arises obliquely. The coats of the cecum are all found in the appendix. The lumen of the latter is small, except near the entrance, and the walls may be in contact. The lymph nodules of the appendix are exceedingly numerous and large, in places fusing into masses of considerable size, which encroach upon the mucosa and its glands so as to almost reach the free surface.

The vermiform appendix is a long, slender, worm-like diverticulum from the cecum, formed of all the coats of the intestine. The length varies from 1 centimeter (⅓ inch) to 24 centimeters (9⅓ inches), the average being probably about 8.4 centimeters (3¼ inches). The general direction of the appendix is very uncertain; that of the distal half especially being largely a matter of chance. The appendix is attached to the cecum and to neighboring structures by a peritoneal fold. In the majority of instances the appendix is wholly behind the cecum, mesial to it, or below it.

The artery supplying the cecum is the ileocolic, a branch of the superior mesenteric, which sends to it both an anterior and a larger posterior branch, which ramify downward over the front and back of the cecum. The artery of the vermiform appendix arises from the posterior division of the ileocolic, crosses the back of the ileum, and runs in the fold of peritoneum to the end of the appendix. The veins of the cecum are arranged on much the same plan as the arteries.

The lymphatics are divided into a posterior and an anterior set. The former empty into small nodes on the back of the cecum beneath its peritoneal covering. The anterior ones are in or near the fold between the cecum and the colon. The nerves supplying the cecum are derived from the superior mesenteric plexus.

The ascending colon extends from the cecum to the under side of the liver, where it makes a sudden bend—the hepatic flexure—and becomes the transverse colon, which crosses the abdomen to the splenic flexure at the spleen, whence, as the descending colon, it passes to the crest of the ileum. From that point to the middle of the third sacral vertebra it is known as the sigmoid flexure. There are three bands of the colon, formed by accumulations of longitudinal fibers, each of which is about 1 centimeter broad. Their disposition in the walls of the gut is not constant. In the ascending colon one is in front and two behind, one of the latter being near the outer and the other near the inner aspect. In the transverse colon the anterior becomes the inferior, while the external becomes the superior, receiving the attachment of the transverse mesocolon. In the descending colon they assume their original position, but tend to grow indistinct. They are still more so in the sigmoid flexure, and before the rectum is reached there are but two bands, an anterior and a posterior, of which the latter is the stronger. The interior of the colon shows the sacculated condition, but there are no folds or valvulæ conniventes like those of the small intestine. The solitary lymph-nodules continue, much like those of the jejuno-ileum.

The ascending colon mounts in the right flank to the hepatic flexure, which makes a large impression on the under side of the right lobe of the liver directly anterior to the kidney. The transverse colon is suspended between its beginning, the hepatic flexure, and its end, the splenic flexure, like a festoon running forward and downward; for the ends are near the back of the abdominal wall. The splenic flexure is in front of the lower part of the spleen, and is both higher and more posterior than the hepatic one.

The descending colon descends from the splenic flexure and generally is much more contracted and sacculated than the first part. The sigmoid flexure begins at the crest of the ileum as a loop of varying length, which is attached by a mesentery. Its usual length is from 25 to 56 centimeters (10 to 18 inches) but occasionally is longer. Sometimes it is quite straight, at other times it is very much convoluted, and may assume almost any shape.

The lower part of the ascending colon is very often, for one or two inches, completely surrounded by serous membrane. The ligaments of the colon are more or less well marked at the line where the peritoneum leaves the posterior wall. The transverse colon is attached to the transverse mesocolon and is completely surrounded by peritoneum. The length of the colon between the cecum and the transverse is only covered in front by peritoneum. It is therefore

extra-peritoneal. The descending colon is likewise uncovered posteriorly by peritoneum. At the sigmoid flexure the peritoneum usually surrounds the gut. The rectum proper has no peritoneal covering.

THE RECTUM, ANAL CANAL, AND ANUS.

The rectum begins at the middle of the third sacral vertebra, the point at which usually the mesentery that restrains the sigmoid flexure terminates. The rectum descends along the hollow of the sacrum and coccyx, passes the point of the latter, and continues until

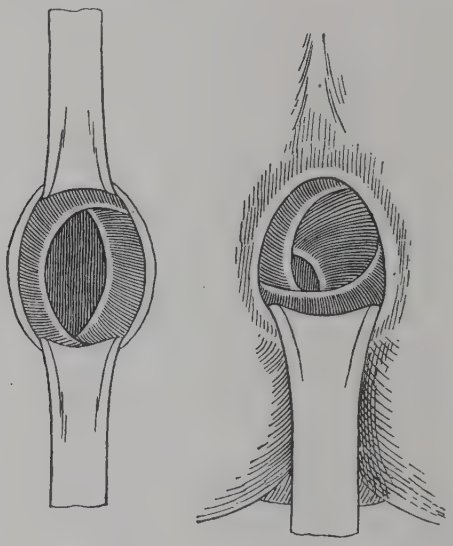

Fig. 6.—Folds of the rectum seen after dilation. (*Otis.*)

it reaches the lower and back part of the prostate gland in the male or the vagina in the female. Its length is approximately 12.5 centimeters or 5 inches. The gut is then continued by the anal canal, sometimes called the sphincteric portion of the rectum, situated in the thickness of the pelvic floor, and directed downward and backward, making a sharp angle with the rectum proper.

The rectum proper, having passed the tip of the coccyx, rests on the levator ani muscle, although separated from it, as well as from the sacrum and coccyx, by the dense rectal fascia. The rectum is sacculated, presenting, when distended, usually three dilatations, of which the lowest and largest, called the ampulla, may measure 25 centimeters (9⅞ inches) or more in circumference. The saccules are separated by deep creases, passing about two-thirds around the

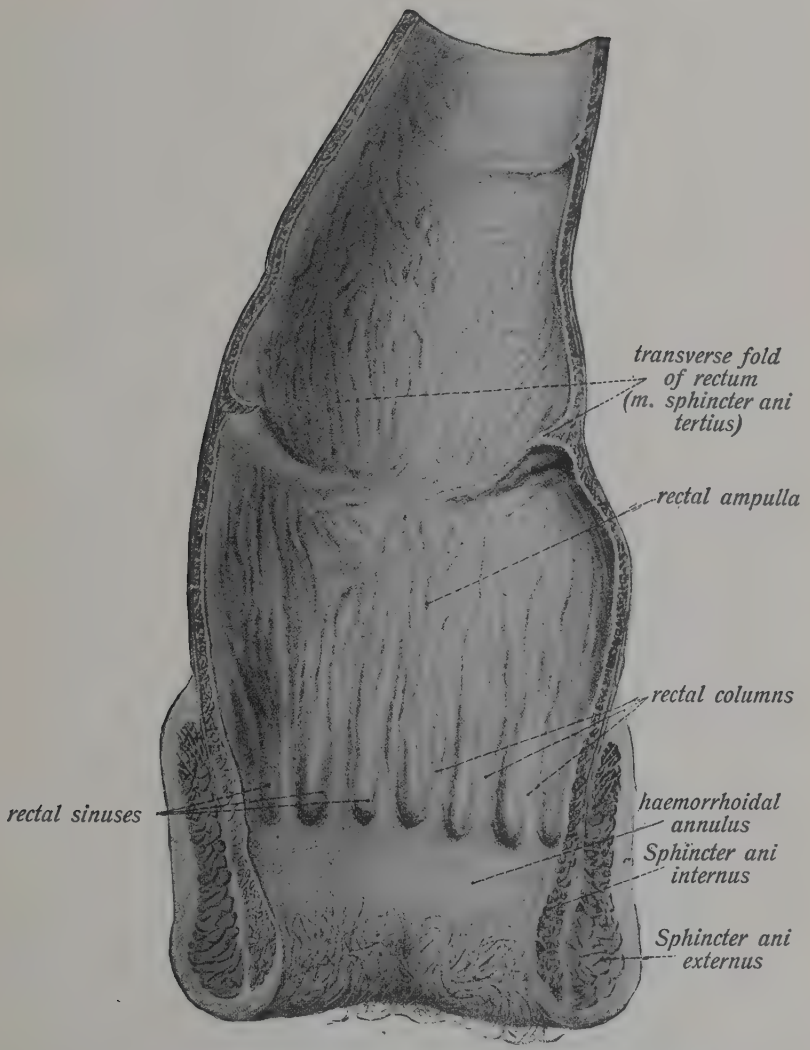

Fig. 7.—The submucous membrane of the rectum and anus. (*Sobotta-McMurrich.*)

gut, caused by a folding-in of the coats internal to the two bands of longitudinal fibers. These folds form the so-called valves of the rectum.

THE ANAL CANAL.

The anal canal is situated in the thickness of the pelvic floor and extends downward and backward. It differs from the rest of the intestinal canal in having no lumen under ordinary circumstances, when the sphincters surrounding it are contracted. The anus is a term used to denote the termination of the anal canal. It is deeply situated between the nates, especially in the female. Anatomically it is the lower end of the anal canal, since the skin which is puckered up by the external sphincter and the corrugator cutis ani, sometimes resembles mucous membrane, so that the anal canal appears really longer than it is. The anatomical boundary, the anal-rectal groove, or the so-called white line of Hilton, is a slight zigzag furrow, usually to be seen in the living, and not in the dead. It lies a little above the lower limit of the internal sphincter, which, covered by dilated veins, projects toward the potential lumen above the external sphincter. The moist and dark skin which is puckered up to form the continuation of the anal canal is very thin, but gradually assumes the appearance of ordinary integument. The so-called anal glands surrounding the anus are of two kinds, both of which have their orifices in this skin. Those nearest to the boundary line are sebaceous follicles, and external to them is a zone of large sweat glands.

The mucous coat of the rectum is thick and vascular, and corresponds in histological details with the mucosa of other parts of the large intestine. The glands of Lieberkühn are exceptionally large, and the muscularis mucosæ is better developed than in the colon. The rectal valves are two or three folds, exceptionally four or five, projecting like transverse shelves into the cavity when it is distended, and hanging loose when it is not. They are semilunar in shape, with the greatest breadth from the attached border to the free edge, ranging from 1 centimeter to more than 3 centimeters. They contain all the coats of the gut, except that, chiefly on the posterior wall, some of the longitudinal muscle-fibers pass outside of them, thus securing the fold. In the large folds there is an accumulation of the circular fibers. These folds tend to be effaced in the opened rectum, are placed laterally, and have in common that their points cross the middle line, although not symmetrically, extending more onto the front than the back.

The columns of Morgagni are a series of permanent vertical folds of mucous membrane passing from the anal canal up into the rectum. The number of these folds varies from five to considerably more than ten, which latter number is perhaps about the average. The valves of Morgagni are semilunar folds of the mucous membrane connecting the bases of the columns of the same name, and forming with them a number of pouches opening upward. They are situated in the anal canal at the upper part of the internal sphincter. The mucous

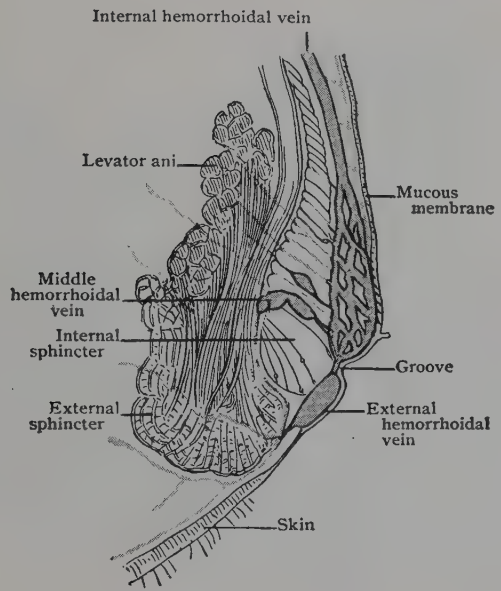

Fig. 8.—Veins within the internal sphincter of the anus. (*Otis.*)

membrane of the rectum is thrown into a series of longitudinal folds. These are easily effaceable, although some are continuous with the columns of Morgagni.

The submucous coat of the rectum is lax, allowing the mucous membrane to be readily displaced, but at the lower end of the anal canal the latter is firmly attached to the muscles. The muscular coat of the rectum is thicker than that of the colon, reaching to 2 millimeters. The thickening is greatest in the layer of circular fibers. The internal sphincter is but an hypertrophy of the circular muscles, while the external sphincter is a muscle of the perineum.

The posterior surface of the highest part of the rectum is usually coated like the rest with peritoneum, except near the median line; below this the posterior surface is without serous covering, and rests

in a dense rectal fascia. The sides and front of the rectum are covered with peritoneum, which is reflected laterally.

The arteries supplying the rectum are derived chiefly from the three hemorrhoidals, the superior, middle and inferior. The general distribution of the veins does not differ much from that of the arteries. The superior hemorrhoidal veins, tributaries of the inferior mesenteric, drain into the portal system. They form a rich plexus throughout the rectum, particularly in the upper and middle parts of the anal canal. In this situation they present a series of dilatations, encircling the gut at the bases of the columns of Morgagni, just above the boundary line between the mucous and cutaneous areas. The lymphatics pass to the sacral glands in the front of the rectum. In the lower part a very rich plexus is found under the skin around the anus, which drains into the superior internal inguinal glands. The nerve-supply of the rectum includes both sympathetic and cerebrospinal fibers.

THE LIVER, GALL-BLADDER, AND BILIARY DUCT.

The liver is the largest gland in the body, and is formed of very delicate tissue disposed around the ramifications of the portal vein. Its peculiar shape is chiefly due to the pressure of surrounding organs, its tissue being so plastic that it is molded by them. In the adult it becomes firmer from the increase of connective tissue. It is thickest on the right side and grows thinner toward the left. The greatest diameter is transverse, and the next, vertical. The liver is usually described as composed of five lobes—namely, the right, the left, the lobe of Spigelius, the quadrate, and the caudate. More properly it consists of a right and a left lobe, separated on the superior surface by the falciform ligament. The other lobes are subdivisions of the right lobe, the lobe of Spigelius being at the back and the other two below. The transverse diameter usually nearly equals that of the cavity of the abdomen, although it often falls an inch or so short of it. It may be given at from 22 to 24 centimeters (8½ to 9½ inches). The greatest vertical dimension or depth is about 16 centimeters (6¼ inches); the antero-posterior diameter 12 to 18.5 centimeters (4¾ to 7¼ inches). The weight is, with considerable variations, generally from 1450 to 1750 grams, or approximately from 3 to 3¾ pounds and in the adult is about one-fortieth of the body weight. The color is a reddish brown, and the naked eye can recognize that the surface is covered with the outlines of polygons from 1 to 2 millimeters in diameter. These are the lobules,

each of which is surrounded by vessels and ducts in connective tissue, and contain in the middle a vessel, the beginning of the system of the hepatic vein. The superior surface is in the main convex, looking upward beneath the diaphragm. The inferior surface is molded over the organs beneath it and is essentially concave.

The most important surface for the purposes of this volume is the inferior. This is subdivided by a system of fissures which may be

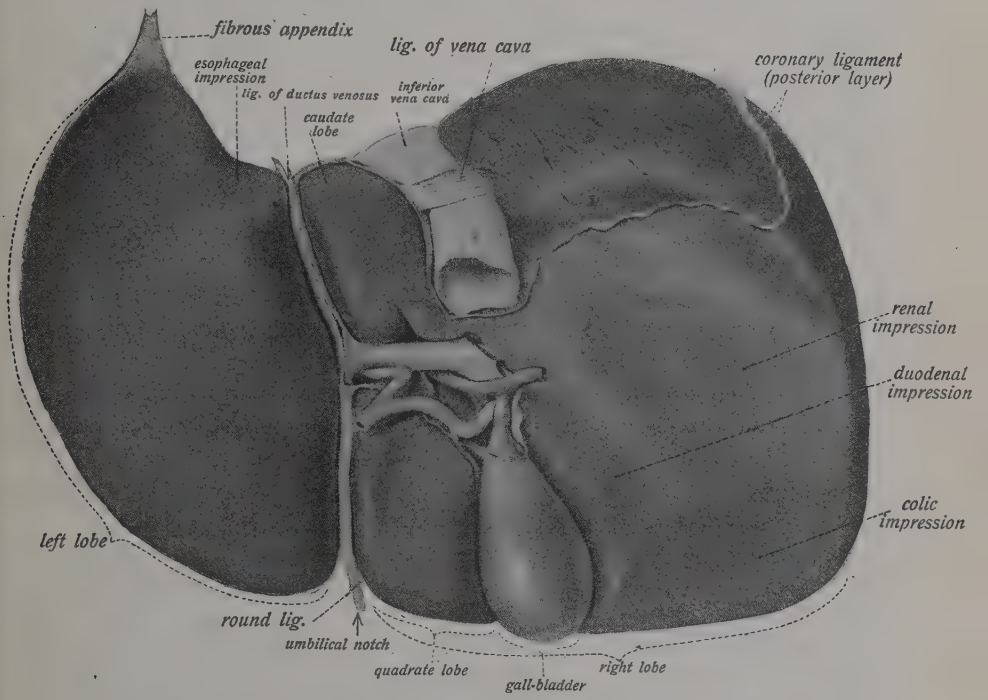

Fig. 9.—The under surface of the liver. (*Sobotta-McMurrich.*)

described as resembling the letter H, in which the cross-piece—the portal fissure—is not in the middle, but very near the posterior border. The portal or transverse fissure is of an entirely different nature from the others. It is the hilum of the organ for the passage of the vessels and ducts; while the other fissures more properly deserve the name, being due to the pressure of the gall bladder and the vessels. The portal fissure is from 4 to 5 centimeters (1½ to 3 inches) long. It transmits the portal vein, the hepatic artery, the subdivisions of the gall-duct, the lymphatics and the nerves, all enveloped in a mass of areolar tissue known as Glisson's capsule. The large portal vein is posterior. The hepatic artery lies in front of

it on the left and the hepatic duct, formed by the two chief tributaries, lies before it on the right. The lesser omentum is attached to the lips of the fissures outside of these structures. At the left end the portal fissure receives the umbilical fissure, which runs backward from the notch in the anterior border and contains the obliterated umbilical vein, in the adult this is known as the round ligament. This fissure is very often bridged over. Continuous with the umbilical fissure, the fissure of the ductus venosus ascends the posterior surface, only a small part of it being on the inferior aspect. At the left end of the portal fissure the falciform ligament joins the lesser omentum, the latter being continued backward in the fissure of the ductus venosus. The fossa for the gall-bladder is a depression on the under surface of the right lobe, in which that organ rests. Resting on the under surface is the liver, the gall-bladder, the right kidney, the suprarenal capsule of which rests against the liver, the first part of the duodenum, and the hepatic flexure of the colon.

The portal vein, some 15 millimeters or more in diameter, divides into a right and a left branch, 10 millimeters or over in diameter, of which the right is a little the larger and shorter. From the right end of the transverse fissure it runs backward in a curve to the right of the vena cava, keeping in the lower part of the liver and giving off successively a series of large branches to the front and right of that organ. The portal vein splits up into a number of branches which go into various parts of the under surface of the liver.

The hepatic veins carrying off the blood from the liver arise as the intralobular veins, which empty into the sublobular, which in turn join larger vessels converging towards the vena cava. The arrangement of the hepatic branches in the main is like that of the portal, but near the edge of the liver we find more instances of the union of two rather small trunks meeting symmetrically like the arms of a Y. The ramifications of the portal and hepatic veins are inextricably mixed throughout, but in the main the branches of the latter lie above those of the former. The hepatic artery, the nutritive vessel of the liver, divides into two branches which, together with the bile duct, accompanying the portal vein, the two arteries generally being on the same side of the vein. The lymphatics of the liver constitute a superficial and a deep set, the former lying beneath the peritoneum, the latter within the deeper interlobular connective tissue. The superficial lymphatics of the superior surface are arranged as three groups, posterior, anterior and superior. The deep lymphatics include two distinct groups, the one following the branches of the portal vein, the other accompanying the hepatic veins. The first descends

within the capsule of Glisson in company with the portal vein and the other interlobular vessels. Below the hilum of the liver they empty into a series of hepatic nodes.

The nerves are chiefly derived from the solar plexus of the sympathetic with some fibers from the left pneumogastric which reach the liver by passing from the anterior surface of the stomach between the layers of the lesser omentum.

The glandular tissue composing the liver is subdivided into small cylindrical masses, the lobules, by the connective tissue, which is the continuation of the fibrous envelope, or capsule of Glisson which enters at the transverse fissure and accompanies the interlobular vessels in their ramifications. The distinctness with which the lobules are defined depends upon the amount of this interlobular tissue.

Since the liver is essentially a vascular organ it is desirable to study the vascular distribution before considering the disposition of the hepatic cells. The portal vein, the functional blood-vessel of the organ, ramifies within the capsule of Glisson and finally encircles the periphery of the lobule. Numerous minute branches are given off from the interlobular ramifications of the portal vein which enter the periphery of the adjacent lobules and break up into the intralobular capillary network. The disposition of the latter is in general radial, the capillaries converging towards the middle of the lobule, where they join to form the central or intralobular vein, the beginning of the system of the hepatic veins by which the blood passing into the lobules is eventually carried into the inferior vena cava. Within the lobule is a capillary network, composed of channels with a diameter of about 0.01 millimeter which terminate in a central vein occupying the long axis of the lobule and increasing in size as it proceeds towards the base of the lobule. Immediately upon emerging from the lobule the central vein opens into the sublobular vein, which runs usually at right angles to its intralobular tubularies and along and beneath the bases of the lobules, the outlines of which are often seen through the walls of the vein. The sublobular veins join to form larger vessels, which in turn unite and constitute the branches of the hepatic veins.

In the meshes of the interlobular capillary network are found the hepatic cells, the bile capillaries, and a meager amount of connective tissue. The cells are arranged as cords or trabeculæ which conform in their general disposition to the intercapillary spaces, which they completely fill. In a sense, the entire lobule consists of a solid mass of hepatic cells tunneled by radially coursing capillaries and their short anastomosing branches, the proportion of the space

occupied by the vascular channels to that filled by the cells being approximately as 1 to 3. When isolated, the liver-cells present a polygonal outline and measure usually from 0.015 to 0.025 millimeters in their longest dimension. Each cell usually exhibits a shallow depression which indicates the surface of former contact with a capillary and emphasizes the intimate relation existing between the blood-vessels and the cells. The liver-cells consist of finely granular protoplasm which sometimes exhibits a differentiation into an outer and an inner zone. It is without a cell membrane, although the peripheral zone of its cytoplasm is condensed, especially when it forms part of the wall of the bile-canaliculi. The nucleus, of vesicular form, contains a small amount of chromatin and usually a nucleolus. Occasionally a large cell is met with, as well as one with an unusual diameter of nucleus.

The bile-capillaries, representing the lumina of ordinary tubular glands, form a network of intercommunicating channels throughout the lobule closely related to the liver-cells. While the predominating direction of the bile-capillary is radial and corresponds to the similar general disposition of the cylinders or leaflets of hepatic tissue, the radial arrangement is converted into a network by numerous cross-branches. The diameter of the bile-capillary is from 0.001 to 0.002 millimeters and remains practically the same throughout the lobule until the canaliculi reach the extreme periphery. The intralobular bile-ducts which receive the biliary canals that pierce the periphery of the lobule as the outlets of the intralobular network, accompany the branches of the portal vein and the hepatic artery within the capsule of Glisson. These ducts, from 0.030 to 0.050 millimeters in diameter, constitute a network over the exterior surface of the lobule. They consist of a dense fibro-elastic coat lined with cylindrical epithelium. Beginning as small vessels which surround the lobules, they become contributory to the larger bile-ducts, which increase in diameter as they approach the transverse fissure. In the vicinity of the latter these trunks join into the two main lobular ducts forming the hepatic duct. The largest bile-vessels possess bundles of unstriped muscle which in the hepatic duct are arranged principally as a longitudinal layer, supplemented by circular and oblique bundles.

In addition to the small interlobular bile-vessels already described, the system of canals receiving and conveying the secretion of the liver to the intestinal tract consists of the hepatic duct, the excretory tube of the organ; the gall-bladder, a reservoir in which the bile accumulates during the intervals of digestion; the cystic duct, the continuation of the bile-sac opening into the side of the hepatic

duct; and the common bile-duct, which, although formed by the union of the other two, is in structure and direction really the continuation of the hepatic duct.

The hepatic duct is formed below the hilum by the union of its two—the right and left—chief tributaries. The latter issue from the portal fissure, one on each side, and generally unite with the hepatic duct somewhat in the shape of a T, the last named canal forming almost a right angle with each of its tributaries. The length of the hepatic duct varies, being usually from 20 to 40 millimeters (¾ to 1½ inches), with a diameter of about 4 to 6 millimeters. It lies in the gastro-hepatic omentum, in front of the portal vein and to the right of the hepatic artery, and inclines downward to the inner side of the second part of the duodenum, resting previously on the top of the first part. The hepatic duct ends at the point at which the cystic duct opens into it. The duct is lined with mucous membrane, covered with simple columnar epithelium, and presents many minute pits, into which open the orifices of numerous small tubular glands. Its walls consist of a fibro-elastic tissue and unstriped muscular fibers.

The gall-bladder is a pear-shaped receptacle for the bile, resting in its fossa on the under surface of the liver, with its large end forward. The long axis runs also somewhat inward. The length is from 8 to 10 centimeters (3¼ to 4 inches), and the capacity some 50 cubic centimeters (about 1½ fluidounces). It narrows to a point where it usually bends to the left and ends in the cystic duct without definite external demarcation. The bent terminal portion, or neck, about 1 centimeter long, is more or less closely bound beneath the peritoneum to the side of the gall-bladder, so that before this is separated it sometimes looks as if the duct arose from the side of the latter. The fundus of the gall-bladder lies near the end of the ninth right costal cartilage. The neck is at the right end of the portal fissure. Anteriorly the bladder rests on the transverse colon, behind which it lies first to the right of and then above the first part of the duodenum. The wall of the gall-bladder is very resistant, being composed of a mixture of fibrous tissue and of unstriped muscular fibers. Most of the latter are disposed circularly, but the oblique and longitudinal ones are interwoven. The fibro-muscular tunic is lined by a layer of mucous membrane which is very adherent to it. The mucous membrane is covered by simple columnar epithelium.

The artery of the gall-bladder is the cystic artery, a branch of the hepatic. The superficial veins join the cystic vein and empty into the right division of the portal vein. The lymphatics, for the most part, empty into the nodes in the portal fissure. The nerves

are derived through the solar plexus through the hepatic plexus. The cystic duct is 3 or 4 centimeters in length, with a diameter of about 2 to 3 millimeters and passes in a fold of peritoneum from the neck of the gall-bladder to the gastro-hepatic omentum, where it joins the hepatic duct at an acute angle, or, rather, opens into its side. It is said sometimes to present an enlargement at its end.

The common bile-duct is about 7 centimeters (2¾ inches) long, and its diameter is about 6 to 7 millimeters at the commencement and rather less at the end. Beginning immediately below the transverse fissure, although conventionally regarded as formed by the union of the cystic and the hepatic ducts, being, in fact, the direct continuation of the latter, the common bile-duct passes downward between the layers of the gastro-hepatic omentum, in front of the foramen of Winslow, with the hepatic artery to its left and the portal vein behind. It descends along the postero-inner aspect of the bend joining the first and second parts of the duodenum, then along the inner side of the second part, where it is more or less surrounded by the head of the pancreas. Near its termination it meets the pancreatic duct, and in company with the latter, pierces the duodenal wall, which it traverses obliquely for the distance of some 15 millimeters, to empty into the duodenum at a papilla marking the common orifice of the two ducts.

There are no true ligaments of the liver. They are folds of peritoneum which are essentially five in number. The so-called round ligament, which is a cord of fibrous tissue, the remains of the obliterated umbilical vein, the falciform ligament, a peritoneal fold representing the primary anterior mesentery, the coronary ligament, and the right and left triangular ligaments.

THE PANCREAS.

The pancreas, sometimes known as the abdominal salivary gland, lies molded across the spinal column with its head on the right, enclosed in the loop of the duodenum, and its tail on the left, in contact with the spleen. It is of a light straw color running into red, according to the amount of blood within the organ. The weight ranges from 30 to 150 grams (1 to 5 ounces) or more. The length *in situ* is approximately 15 centimeters (about 6 inches). It consists of an enlarged descending part on the right, the head, and of a long body placed transversely which is needlessly divided into neck, body and tail.

While agreeing in general structure with other serous salivary glands, as the parotid, the pancreas differs in certain particulars.

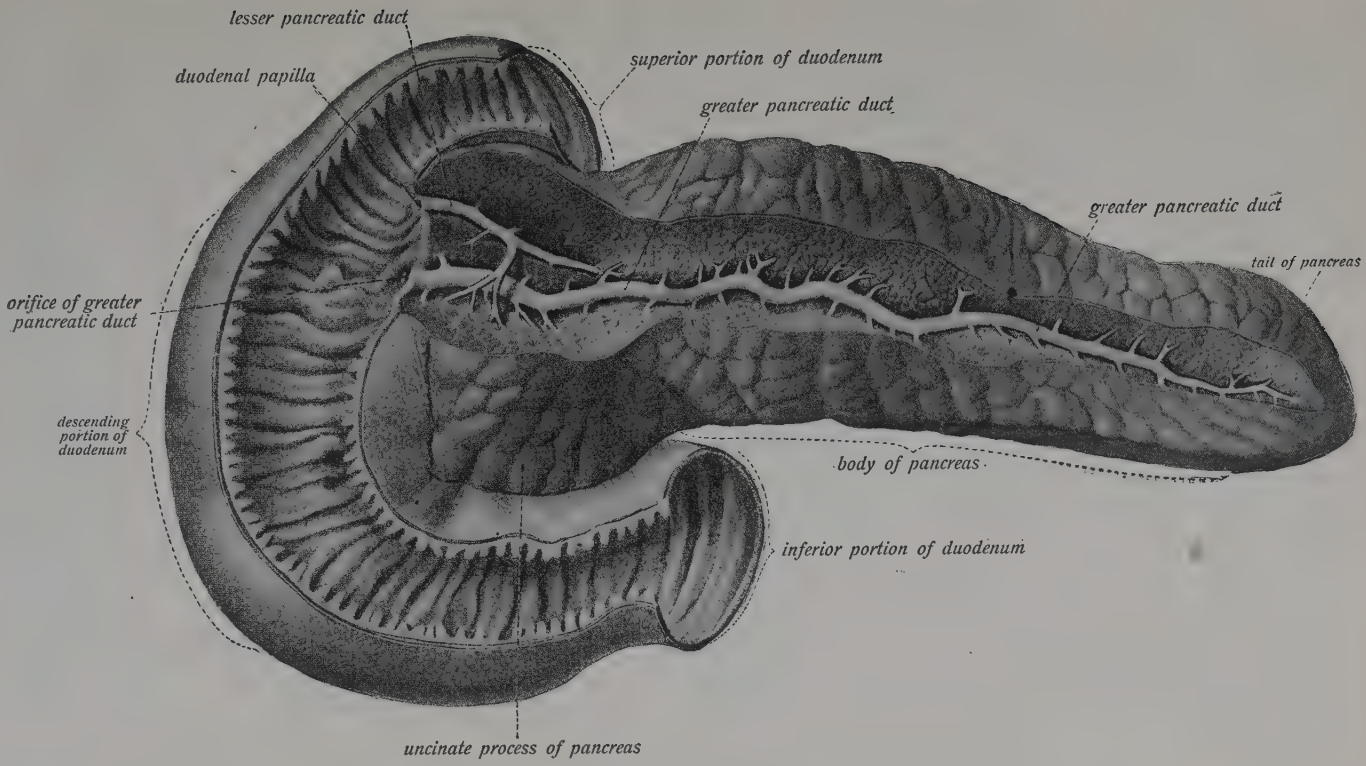

Fig. 10.—Pancreas, ducts and duodenum. (*Sobotta-McMurrich.*)

Most important of these are the tubular rather than saccular form of the alveoli, the marked differentiation of a granular zone in the protoplasm of the secreting cells, the absence of specialized intralobular ducts, and the presence of the islands of Langerhans.

The chief pancreatic duct gives off numerous lateral interlobular branches which are lined with a single layer of columnar epithelium. The canals springing from the interlobular ducts after entering the lobules possess a layer of flattened epithelial plates.

The tubular alveoli of the gland, often tortuous and sometimes divided, possess a well-defined membrana propria against which lie the secreting cells. The latter are usually of a blunted pyramidal shape, with an average length of about 0.010 millimeters. During functional inactivity their cytoplasm exhibits two well-differentiated zones; an inner one, next the lumen, which is highly granular, and an outer one, next the basement membrane, which is free from granules and at times almost homogeneous. A round or oval nucleus occupies the external area. During fasting, when cells are stored with zymogen particles, the granular zone is very broad and the outer homogeneous one correspondingly narrow. With the beginning of discharge of the pancreatic secretion during digestion, the granular zone diminishes and reaches its minimum, almost disappearing when the gland is exhausted. When function is resumed there has been a formation and gradual accumulation of a new store of zymogen particles in the granular zone.

The interalveolar cell areas, known as the islands of Langerhans, appear as small collections of cells, some 0.3 millimeters in diameter, lying between the tubular acini, from which they are separated by a delicate envelope of connective tissue. These cell areas are constant features of the pancreas, not only in man, but likewise in varying types of animals. Their distribution within the pancreas is not uniform. The cells composing these masses differ from the elements of the pancreas in being smaller, polygonal rather than pyramidal in form, less granular, and undifferentiated into the characteristic zones usually seen in the pancreatic cells. They are arranged as a network, consisting of solid cords, the meshes of which are occupied by blood-capillaries of large size; the whole recalling the arrangement of the hepatic tissue. It is assumed that these cells are largely concerned in internal secretion.

The pancreatic duct, the duct of Wirsung, begins near the end of the tail and runs through the middle of the pancreas toward the right, and bends downward as it passes through the head. Branches sprout from the main duct at right angles, which receive bundles

of smaller ramifications. The diameter of the duct near its end is about 5 millimeters. It descends just in front of the common bile-duct to the wall of the duodenum and empties in common with it at the papilla. The tributary ducts of the head are sometimes rather numerous. A particularly large one, the duct of Santorini, is the remains of the early stage of development and empties into the duct of Wirsung as the latter turns downward.

The arteries of the pancreas are many small branches derived from the splenic, hepatic and superior mesenteric. The veins follow the main artery. They are all tributary to the portal system, and some open directly into the portal vein. The lymphatics are many. Most of them run to the celiac and splenic plexuses. A group of small lymph-nodes is situated in the front of the head. The nerves, composed chiefly of non-medullated fibers, are from the solar plexus, by way of the celiac, splenic, and superior mesenteric plexuses.

THE PERITONEUM.

The peritoneum is the serous membrane lining the abdominal cavity and reflected over the viscera. Like all serous membranes, it consists of a free mesothelial surface, and a deeper layer of fibro-elastic tissue, the tunica propria. Beneath the latter a variable amount of subperitoneal tissue connects the peritoneum with the structures which it covers. The quantity of this areolar layer differs in various locations, and it is sometimes difficult to decide just what is really a part of the serous membrane proper. It serves to connect the peritoneum with the various viscera and with the abdominal wall. The anatomy of the peritoneum is so complicated and complex that the author considers it unwise to describe it in detail in this volume and refers the reader to works on anatomy.

CHAPTER II.

Physiology and Chemistry of Intestinal Digestion.

THE products of gastric digestion, after being worked up in the pyloric half of the stomach, are passed at intervals into the first part of the duodenum. From here downward the major part of digestion takes place, the contents being acted upon by the pancreas, the liver and the tubular glands of the intestines, the secretions of which are discharged into the gut. There is also a secretion from the glands of Brunner which are situated at the beginning of the duodenum. The secretion of these various juices is practically simultaneous and is aroused by the entrance of the acid chyme into the duodenum. While they operate in conjunction, it will be convenient to deal separately with the various secretions in regard to their action and mechanism.

PANCREATIC JUICE.

This is a clear, slightly opalescent fluid, strongly alkaline from the presence of sodium carbonate. Its alkalinity about equals in degree the acidity of the gastric juice, a provision of nature probably intended to keep a constant running neutrality in the contents of the intestinal canal. According to Starling, the proteins of the juice may be roughly divided into three groups, a small amount of nucleo-protein precipitated on acidification, a protein coagulating at 55°C., and another one at about 75°C. As the pancreatic juice is discharged it tends to become poorer in proteins and richer in alkali. The most interesting and important constituents of the juice are its ferments or the precursors of ferments. The juice on reaching the intestine has, or develops, an effect on all three classes of food-stuffs, namely: proteins, fats, and carbohydrates.

It is definitely known that pancreatic juice is secreted without the power of changing protein substances, and that this effect is brought about by some change in the juice itself, namely, a conversion of trypsinogen into trypsin, the ferment which is active in digestion. This change under normal circumstances is brought about directly the juice enters the gut, by the action of a substance known as

enterokinase which is contained in the succus entericus. In the presence of this product, the pancreatic juice acquires a proteolytic activity superior to that of any other digestive juice, so that all the normal proteins of the food undergo a very thorough disintegration. Because of the various constituents in their make-up, foods are broken down into substances known as amino-acids, and the same change is undergone by the proteoses and peptones resulting from the gastric digestion of proteins. Within a few minutes after the chyme has reached the small intestine certain amounts of amino-acid are formed. A portion, however, is not formed until late, showing a resistance of some proteins to disintegration. After tryptic digestion for a few hours the mixture will be found to contain a considerable quantity of peptone, which in consequence of its resistance to further alteration was designated by Kühne "antipeptone." The antipeptone of Kühne certainly included some of the diamino-acid which at that time had not been isolated. There is always a part, however, which gives the biuret reaction and is, after prolonged action of trypsin, only slightly broken down into the amino-acids. Among the amino-acids, tyrosine is one of the first to be split off, and this substance, with leucine, was among the earliest known products of pancreatic digestion.

Since the pancreatic juice is strongly alkaline, it might be expected that complete protein reduction would be only accomplished in an alkaline medium. But it is interesting that the action of trypsin is seen to closely resemble the action of the boiling of concentrated hydrochloric acid. Like the latter, it affects the protein molecule at the CO-NH coupling introducing water at this point, and therefore breaking up the polypeptid groupings into simple amino-acids. However, a neutrality exists throughout the small intestines, the reaction of the contents of the gut being similar to that of a fluid containing alkali which has been saturated by the passage of carbonic acid, that is, alkaline to the well-known indicator of methyl-orange and acid to phenolphthalein.

Trypsin is found to be most active in the presence of sodium carbonate. It is probable that although an alkaline reaction is most advantageous for the earlier stages of tryptic activity, the later stages take place in a neutral medium. This is probably due to the fact that trypsin in an alkaline medium is extremely unstable and that when prolonged digestion is necessary it would be destroyed if the medium were strongly alkaline. The destructability of trypsin, as well as its action, is largely affected by the presence of protein or its digestion products in solution. The more peptones or amino-acids

present the less rapid the action of the trypsin, and it has been found that if by any means the amino-acids are removed, the action of trypsin is renewed. It is probable that this destruction of ferment is constantly taking place in the intestine itself. The tryptic effect of the pancreatic secretion on chyme is most active in the upper part of the gut, and generally is destroyed before reaching the lower end. As was mentioned before, the pancreatic juice possesses no proteolytic activity, but that a ferment named enterokinase is needed in the duodenum to convert the trypsinogen into active trypsin. Parlow therefore called enterokinase the "ferment of ferments." It has been suggested that a combination takes place between the enterokinase and the trypsinogen, trypsin itself being a mixture or combination of the two bodies. This has been disputed by Bayliss and Starling on the basis that an equality of proportion is not present, and that the smallest quantity of enterokinase is capable of activating large amounts of trypsinogen.

The action of pancreatic juice upon milk is to produce a clot which is speedily redissolved. In this it is unlike the action of the gastric juice upon milk wherein a clot is produced which remains for some time. It can be proven artificially, that sometimes the clot forms so quickly, and the redissolving so soon afterward that the production of the clot is missed. While it is possible that a rennet-like ferment is in the juice, such as has been considered as present in the gastric juice, it is more probable, as mentioned in my text-book on the stomach, that this clotting and redissolving is a first stage in proteolysis, and that no rennet-ferment is necessary therefor.

The pancreatic juice, as well as the fresh extract of the glands, contains a strong amylolytic ferment known as diastase, amylase, or amylopsin. Its effect on starch is extremely rapid. At the end of a few minutes no color is obtained with iodine, and the solution contains maltase. The action in the hydrolysis of starch is exactly the same as that brought about by ptyalin, excepting more active. When the juice is neutralized the process of hydrolysis goes on to the formation of dextrose or glucose. A further conversion takes place in the presence of a second ferment—maltase—which converts the disaccharide maltose into the monosaccharide glucose. In this way when the digestion of sugar or the starches is left off in the mouth it is finished by the pancreatic juice. On the other disaccharides the pancreatic juice is without effect. It therefore has no effect upon cane sugar or milk sugar.

Fresh pancreatic juice contains a strong lipase or fat-splitting ferment by means of which in the presence of water, neutral fats—

that is, the triglycerides of palmitic, stearic and oleic acids—are broken up into glycerin and the corresponding fatty acids. For the action of this it makes but little difference whether the ferment is working in an alkaline, neutral or slightly acid reaction. Probably in one strongly acid it is inhibited. This ferment is soluble in glycerin but not in distilled water. The velocity of the action of lipase is enhanced four or five times by the addition of bile. Its action on the part of bile is due entirely to the biliary salts which act in two ways. In the first place by the physical quality they diminish the surface tension between the water and oil, and in that way a closer contact may be expected between the watery solution contained in the juice and the oil which is presented to it. In the second place, bile salts have a solvent action on soaps as well as on fatty acids in slightly acid medium. It can therefore be seen that bile is a favorable excipient or medium for the interreaction of the lipase and the neutral fats. The lipase of the pancreatic juice will also hydrolyze the esters of the fatty acids. In the fasting condition there is as a rule no secretion of pancreatic juice. In this the secretion is unlike that of the gastric juice which is more or less constant even when there is no food in the stomach. Within a very short time after the ingestion of food, not more than a minute or two, the flow of pancreatic juice takes place, continuing for two or three hours after a meal, then gradually diminishing. As if in order to meet the rapid exit from the stomach, promptly at the moment that chyme begins to be ejected into the duodenum a large flow of pancreatic juice is taking place. The stimulant for this is in all probability the HCl secretion in the stomach, because alkalies do not produce any pancreatic juice secretion. It is necessary, however, for the acids to come in contact with the duodenal mucous membrane. What probably takes place shortly after the ingestion of any meal is an acid output from the stomach, that is, it is probable that before the real gastric digestion takes place there is some exodus into the duodenum of acid which acts as an excitor of the pancreatic secretion. The flow beginning, it is probable that the action of the nerves plus the alteration in the blood supply in the gland itself, to which the gland is very susceptible, continues its secretion. But it is more probable that the two nerves, namely the splanchnic and the vagi, are also constantly being acted upon by the introduction of acids into the duodenum. Bayliss and Starling found that a secretion of juice was produced even when the acids were introduced into a loop in the small intestine entirely free from any possible nervous connection with the rest of the body. They concluded on the basis of this that the acids produced some

product of the epithelial cells which was absorbed in the blood stream and was the active agent in exciting the pancreatic secretion. To this they gave the name "secretin" or "pancreatic secretin" to distinguish it from possible other members of the same class. They claim further that it is produced in the mucous membrane from a precursor —prosecretin, the latter not having been isolated. Accepting secretin as the activator of the pancreas we have a striking example of a correlation between the activities of two different portions of the body affected by chemical means—hence, secretin is not a ferment. The acid chyme enters the first part of the duodenum and immediately a certain amount of secretin is produced by the acid in the cells of the mucous membrane. This secretin is carried by the blood stream to the cells of the pancreas exciting the secretion of the pancreatic juice. As long as the stomach contains food there is a constant renewal of this local reflex mechanism. While the content of the duodenum is acid the pylorus remains firmly closed. As soon as enough pancreatic juice has neutralized the contents of the duodenum, an exit of chyme from the stomach takes place which again acidifies the mucous membrane in the duodenum with the production of secretin and so on. In this way the whole chain of process goes on until the stomach is empty and all of its contents have passed into the intestines.

THE LIVER AND BILE.

It must be expected from the anatomy and the large size of the liver that the organ has little to do with the actual process of digestion. It is instead the great chemical factory of the body receiving by the portal vein the products of digestion as they are absorbed from the alimentary canal. These are converted into other substances, breaking them down and building them up according to the needs of the body as a whole. For instance, glucose contained in the portal veins is converted into glycogen which the liver stores up to be reconverted into glucose for circulation when this substance is required by the body tissues. In the complete absence of carbohydrates, the liver has the power to convert the products of protein digestion into sugar. It also plays a considerable rôle in the metabolism of proteins and of fats.

What concerns us most, however, is the function of the liver in connection with digestion—that is, its function as a secreting gland. The connection of the biliary duct with the pancreatic duct, both opening into a common orifice, shows that the secretion of the bile and the pancreatic juice has a combined action. Firstly, the bile must

be regarded as an excretion because it is the channel by which the products of disintegration of hemoglobin are gotten rid of from the organism. Bile is required in the gut only during the period that digestion is going on. The secretion of bile therefore, like the pancreatic juice, is a discontinuous entry of this fluid into the small intestine. It has been proven that bile which has been collected in the gall-bladder is somewhat different in its composition from that obtained by way of a biliary fistula. Bile obtained from the gall bladder is in every constituent very much heavier than that obtained from a biliary fistula. This therefore shows that during the stay of bile in the gall-bladder it becomes concentrated by the loss of water and by the addition to it of mucin or nucleo-albumin, derived from the cells lining the bladder. While the secretion of bile into the contents of the intestines is intermittent, the manufacture of it by the liver is continuous, and what is then manufactured in the intervals of digestion is collected in the gall-bladder for use during the active period of digestion, when it is quickly discharged into the contents of the gut. It is probable that an increased secretion of bile takes place during the period of digestion, and that this is influenced somewhat by the passage of acid chyme through the pylorus.

Bile contains a weak amylolytic ferment. Its uses in digestion, however, are not dependent upon the presence of this ferment but on the peculiar action of the bile salts on the fermentative powers of the pancreatic juice, and the absorption of fats. The fat-splitting action of pancreatic juice is trebled by the addition of bile, whether the bile be boiled or unboiled. This quickening action of bile probably depends upon the physical properties of bile salts with those of lecithin and cholesterin which they hold in solution. Such a solution has the power of dissolving fatty acids and soap, including even the insoluble calcium and magnesium soap, and no doubt it hastens the effect of the lipase of the pancreatic juice. Therefore the digestive function of the bile lies in its power of serving as a vehicle for the suspension and solution of the interacting fats, fatty acids, and fat-splitting ferments. The vehicular function plays an important part in the absorption of the fats. These pass through the striated basilar membrane bounding the intestinal side of the epithelium in a state of fine emulsion, dissolved in the bile in the form of fatty acids or soap and glycerin. On the arrival of these in the epithelial cells, a process of resynthesis is set up. Neutral fat then makes its appearance, this being passed gradually into the central vessels of the villus and so into the lymphatics of the mesentery and the thoracic duct.

The bile salts are then absorbed by the blood circulating through the capillaries of the villus and carried back by the portal vein to the liver. On arrival in the liver substance they are turned back into the bile. Thus it is that most of the biliary salts are constantly in circulation, by the content in the bowel going back to the liver and out again into the bowel, and very little of it is actually destroyed, or lost in the alimentary canal. That which is destroyed is by the action of organisms met with in the intestine.

INTESTINAL JUICE.

The intestinal juice obtained from a permanent fistula has a specific gravity of about 1010. It is generally turbid from the presence of migrated leucocytes and disintegrated epithelial cells. It contains about 1.5 per cent. of total acids of which about one-half are inorganic and consist chiefly of sodium carbonate and sodium chloride. It is alkaline in reaction, but less so than the pancreatic juice. It contains a small amount of serum albumin and serum globulin, and includes a number of ferments, all of which are adapted to complete the process of digestion of the foodstuffs commencing in the stomach and the duodenum. Of these ferments two are important as concerned in proteolysis. Enterokinase has already been mentioned. This it will be recalled possesses no action itself upon the protein but is necessary for the development of the proteolytic powers of the pancreatic juice. In addition to this, Cohnheim has described a ferment under the name of "erepsin". This possesses no power of digesting coagulated protein or gelatin, and only slowly dissolves caseinogen and fibrin. It has a rapid hydrolytic effect on the first products of proteolysis, converting albumoses and peptones into amino- and diamino- acids, their ultimate cleavage products. Another ferment contained in the intestinal juice is connected with the digestion of carbohydrates. This is called "invertase" which transforms cane sugar into glucose and levulose or fructose, and finally maltase which converts maltose into glucose. In the young, and also in those upon a milk diet, the intestinal mucous membrane contains lactase, a ferment converting milk sugar into galactose and glucose. By means of these three ferments, namely, invertase, maltase and lactase, the digestion of the starches by the amylase of the saliva and the pancreatic juice makes natural provision for all the carbohydrate foods of the animal to be converted into a hexose, in which form alone carbohydrates can be taken up and assimilated by the cells of the body.

In the interaction between the pancreatic juice and bile the intestinal juice possesses an important function. It has an activating power on the pancreatic juice and has numerous other functions to discharge in the digestion of the foodstuffs. There is much difference in the intestinal juice secreted from various levels of the gut to the ileocecal valve. In the upper portion of the small intestine the processes of secretion are the greater while the process of absorption predominates in the lower section—that is, in the ileum. While the process of secretion of the intestinal juice seems presided over by the exhibition of content within the gut, it is probable that in the accomplishment of intermission the central nervous system is a strong factor. Much pertaining to the individual factors of the intestinal juice is difficult of research, because of the ejection of pancreatic juice at about the same time and level. It is probable, however, that the succus entericus, as is the case with regard to the pancreatic juice is presided over by the same mechanism as has been described in connection with the pancreas, namely, the activity of the secretin produced in the intestinal mucosa. However, the glands of the small intestine can be excited by direct mechanical stimulation of the mucous membrane, and in that way a moderate quantity of succus entericus is secreted. It is probable, that in the process of digestion such represents only the minor part of the activation of the secretion of the juice.

FUNCTION OF THE LARGE INTESTINE.

Great difference is found in the structure of the large intestine in different animals. In the carnivorous the large intestine is short and narrow, and possesses little or no cecum; in the herbivora the large intestine is well developed with sacculated walls, and the cecum, that part of the large gut distal to the opening of the ileum into the colon, is very large. Man possesses an intermediate position between these two classes. It is hardly possible to speak of a secretion by the mucous membrane of the large intestine as of importance in digestion. In the herbivora, alkaline carbonate is secreted to neutralize the acid produced in the bacterial fermentation of food but the processes of absorption and secretion keeping pace, there is no accumulation of the products of secretion in the intestine. A secretion of the intestine is that obtained from the typical goblet cells which contain plugs of mucin. This secretion of mucus not only aids the passage of feces along the gut but probably impedes the propagation of the bacteria which are present in countless numbers in the feces. It is just probable that this large secretion of mucin accounts for the difficulty

to cultivate any large numbers of bacteria from feces, most of them being dead. As an absorbing organ the large intestine of man is of little importance. It has been figured on the basis of fistula in man that about 500 cubic centimeters of water passed the ileocecal valve in twenty-four hours. Of these about 400 cubic centimeters undergo absorption in the large intestine. The absorption of food substances, however, in this portion of the gut is very slow and deficient, as compared to the small intestine. Thus it is that feeding by nutrient enemata is always very inadequate. The chief value of the large intestine in carnivorous and civilized man would seem to be as an excretory organ since it plays an important part in the excretion of lime, magnesium, iron and phosphates. In the case of albuminous foods most of them are absorbed before the ileocecal region is reached. But in the case of heavy dieting with vegetables a large amount of vegetable will pass the ileocecal valve, accumulate in the colon and be excreted, usually very much increasing the bulk of feces. Thus it is seen that the large intestine has to do with the absorption of fluids which have passed the ileocecal valve, most of which are in a rather soluble condition. These are absorbed and the mucin is added to the contents of the gut probably for the purpose of inhibiting bacterial action, and the major portion of the colon, probably from the hepatic flexure downward, is concerned mostly in the accumulation and passage of feces outward. The movements of the intestines will be found described in connection with the Roentgenological anatomy and physiology.

THE ABSORPTION OF FOODSTUFFS.

The intake of water, and probably of salts, by the alimentary canal is in accordance with the requirements of the organism as a whole, and is regulated entirely by the central nervous system. This is indicated by the intense thirst experienced in instances of loss of fluid to the body, such as in sweating, diarrhea, hemorrhage, etc. The absorption of water from the stomach may be regarded as *nil*, although from this viscus, alcohol and possibly peptone and sugar, are absorbed to a slight extent. Water or saline fluids introduced pass through the pylorus either without change or are increased by the secretion of fluid from the gastric glands. The chief absorption of water occurs in the small intestine. While the intestinal contents at the ileocecal valve contain relatively nearly as much water as they do at the upper part of the jejunum, absolute bulk is very much smaller so that only a small proportion of the water which is taken

in by mouth remains to be absorbed in the large gut. Water and saline solution are absorbed by the villi in the intestine. These villi increase the absorbing surface of the intestine very much. It has been calculated that each square millimeter of intestine represents an absorbing surface of from three to twelve millimeters. It is believed that the transference of fluid from the contents of the intestine through the villi into the circulation is accomplished by the epithelium which is bounded on its free surface by a "lipoid" membrane—that is, one containing some complex of lecithin and cholesterin and permeable only by such substances as are soluble in lipoids. On the other hand, the cement substance between the cells may be of a different character and possibly permeable to water-soluble substances. It is a well known fact also that water may be excreted by the intestinal mucous membrane. It is also that the absorption of the different foodstuffs, and probably the normal salts of the body, is affected by the cells themselves in accordance with their nutritional needs, and this view is strengthened when we come to examine into the absorption even of normal saline solutions. The absorption of such a solution could be ascribed to the osmotic pressure of the colloids in the blood plasma or lymph within the spaces of the villi. We must conclude that when a fluid is introduced into the intestine an active transference of water from the lumen into the blood stream is effected by the intermediation of forces having their origin in the metabolism of the cells themselves. This work of absorption of the cells may be aided or hindered according to the physical conditions present. If these act against the cells—if the fluid be hypertonic, the absorption is effected more slowly. While with hypotonic solutions the physical conditions concur with the vital activity of the cells in bringing about a very rapid transference of fluid from the gut into the blood vessels.

The absorption of fats is rather simply explained. Fats administered to an animal in excess of its diurnal requirements are stored up in the body in the form in which they are administered. Each cell of the body probably possesses in itself the mechanism for the utilization of these neutral fats, and for effecting their disintegration and oxidation by which they are finally converted into CO_2 and water. The processes of digestion of fat result in the production of glycerin and fatty acids, if the reaction be neutral or slightly acid. If the reaction of the gut be alkaline, the alkali will combine with the fatty acids to produce soaps. If an animal be examined a few hours after the administration of a meal rich in fats, the lymphatics of the intestine are seen to be distended with a milky fluid which is known as

"chyle," and the same fluid is found filling the cisterna lymphatica magna and the thoracic duct. The fat in such chyle may amount to over six per cent. The fat is finally discharged into the general circulation, and why and how it disappears from the general circulation is not known. It is however, utilized by the various tissues of the body and is thus carried to them. Microscopic examination of a section of the villus during fat absorption shows that the absorption occurs for the most part through the epithelial cells, which are closely packed with fat granules. The sum of all that is known about the process of the digestion and absorption of fats, may be said to be the following; Neutral fat is hydrolized into fatty acid and glycerin under the action of the gastric juice, the pancreatic juice, and the succus entericus, the effect of the gastric juice being, however, extremely limited unless the fat be presented to it in a finely divided condition. The lipolytic action of the pancreatic juice and succus entericus is largely aided and increased by the simultaneous presence of bile, which, in virtue of the bile salts lecithin and cholesterin it contains, enables the pancreatic juice to enter into close relation with the fat, and dissolves the products of the activity of the ferment, and so enables it to attack renewed portions of the neutral fat. As the result of this, glycerin is formed (which is soluble in water) and fatty acids or soaps, according as the reaction of the medium is acid or alkaline. The alkaline soaps are soluble in water, the soaps of magnesium and calcium are soluble in bile, free fatty acids are soluble in bile acids. The fat is thus reduced to a condition in which it is soluble in the intestinal contents whatever their reaction. In this state the fat products are taken up by the intestinal mucosa. Within the cells a process of synthesis takes place, the soaps being split and the fatty acids thus set free or absorbed. Being combined with glycerin with the elimination of water it forms neutral fat, which appears as fine granules in the cell protoplasm. These granules are extruded in a somewhat more finely divided form into the intercellular clefts and into the spaces of the villus, whence by the contractions of the musculature of the villus they are forced with the lymph transuding from the capillary blood-vessels into the central lacteal, and thence along the mesenteric lymphatics to the thoracic duct. About 95 per cent. of the fat digested is absorbed, but probably no absorption takes place in the large intestine.

As a result of the action of the various digestive juices all the carbohydrate constituents of the normal diet of man are reduced to the state of monosaccharides. The absorption of these products may take place in any part of the alimentary canal, the greatest phase

in the act of absorption being done by the small intestine. By the time the food has reached the ileocecal valve practically all of the carbohydrate constituents of the food have been absorbed. The majority of physiologists have agreed that carbohydrates gain entrance into the system by way of the portal system. The most striking fact in this connection is the relative impermeability of the intestinal wall to the disaccharides as compared to the monosaccharides. As a body will only take up such quantities of carbohydrates as it requires, the disaccharides are generally useless. Cane sugar or lactose introduced into the blood-vessels or subcutaneously is excreted quantitatively in the urine, and, as might be expected, does not in any way become glycogen of the liver. It is quite different, however, with lactose, glucose, and galactose. It has been found that sugar does not appear in the urine until as much as 320 grams of cane sugar have been ingested, whereas any quantity of glucose over 100 grams may give rise to glycosuria, also that lactose is absorbed still more slowly, and when given in large doses generally gives rise to a diarrhea.

The absorption of proteins probably takes place by the blood-vessels, the protein first being taken up by the epithelial cells. By the influence of the gastric juice, the proteins of the food are dissolved during their stay in the stomach into albumoses and peptones. In the small intestine the process of hydration is carried further, the trypsin of the pancreatic juice carrying the proteins through the stage of secondary albumoses and peptones, and converting them into a mixture of amino-acids and polypeptides. The same end-products result from the action of the erepsin of the intestinal wall on the albumoses and peptones produced by gastric digestion. The digestive juices finally reduce the proteins therefore to a mixture of amino-acids, with a certain remainder of polypeptides consisting of two or three of the amino-acids associated together, which do not undergo further disintegration under the action of the intestinal ferments. We may conclude therefore, that the amino-acids only produced by a protein digestion are absorbed without further change by the blood-stream, that they circulate throughout the body, a certain proportion of them being built up in the tissue in the protein characteristic of that tissue in order to replace the waste caused by wear and tear. The rest, probably the major part of the protein, is taken up by the liver where it undergoes deamination, the nitrogen moiety being rapidly converted into urea and excreted by the kidneys, while the non-nitrogenous moiety is carried to the working tissues to which it serves as a ready and immediate source of energy.

CHAPTER III.

Roentgenological Anatomy and Physiology.

THE examination of the stomach and intestinal canal has become one of the most important spheres of radiographic work, and presents a large factor of diagnosis in the clinical side of the various conditions met with. Notwithstanding the differences in technique and interpretation, it is a satisfaction to know that by means of the X-ray it is possible to ascertain much about the position, size and movements of the stomach, the process of digestion and departures from the normal.

As my work on the stomach takes up the radiographic side of the esophagus and stomach, we may here begin with the small intestine. Before doing so, however, it will be wise to describe the movements of the intestines from a physiological standpoint, and then add the facts as gleaned by X-ray examination. These combined, give a better understanding of the subject than either view alone.

MOVEMENTS OF THE INTESTINE.

Movements of the intestine can be investigated either by observation of the exposed gut or by the X-ray. If the abdomen of an animal which has been carefully treated is opened in a bath of warm normal saline solution so that the disturbing influence of drying and cooling of the gut is excluded, the small intestine will be seen to present two kinds of movements. In the first place all the coils of the gut undergo a swaying movement from side to side, the so-called pendulum movement. These are attended by slight waves of contraction passing rapidly over the surface. Therefore we have the pendulum movement and the circular contraction. The pendulum movements have been carefully studied by Cannon, and Bayliss and Starling, and have been found to consist of the splitting up of the column into a number of equal segments. Within a few seconds each of these segments separates into two portions each half connecting with the corresponding half of the adjacent section. The contractions recur in their original position, dividing the newly-formed segments of contents and re-forming the segments in the

same position which they had at first. Every particle of food is brought successively into intimate contact with the intestinal wall. These movements have no effect on the transfer of the contents downward and a column of food may remain at the same level in the gut for a considerable time.

In the second or circular type of movements, there are two peristaltic motions, one of which involves the contraction of the gut above the fecal food mass and the other the relaxation of the gut below. The contractions above and inhibition below cause an onward movement of the bolus which travels slowly down the whole length of the gut until it passes through the ileocecal opening into the large intestine. The peristaltic contraction involves the co-operation of the nervous system. It is ascribed to the local nervous system contained in Auerbach's plexus, which may be regarded as a lowly organized nervous system with practically one reaction, namely, that known as the "law of the intestines." An anti-peristalsis is never observed in the small intestine. The stimulation of the vagus has the effect of producing an initial inhibition of the whole small intestine, followed by increased irritability and increased contractions. On the other hand, stimulation of the splanchnic nerves causes complete relaxation of both coats of the small gut. It seems, therefore, that the splanchnics normally exercise a tonic inhibitory influence on the intestinal movements, which can be increased by all manner of peripheral stimuli.

As a result of the two sets of movements described above, the food is thoroughly mixed with the digestive juices, and the greater part of the products of digestion are brought into contact with the intestinal wall and absorbed. What is left—a proportion varying in different animals according to the nature of the food—is passed on by occasional peristaltic contractions through the lower end of the ileum into the colon, or large intestine. The lower end of the ileum presents a distinct thickening of the circular muscular coat, forming the ileocolic sphincter. This sphincter relaxes in front of a peristaltic wave and so allows the passage of food into the colon. On the other hand, as a rule it contracts against any regurgitation which might be caused by contractions in the colon. It is interesting in this connection to know that at this area of the gut the action is unaffected by the stimulation of the vagus. The stimulation of the splanchnic, however, which causes complete relaxation of the lower part of the ileum with the rest of the small intestine, produces a strong contraction of the muscle fibers forming the ileocolic sphincter.

Finally, the food reaches the large intestine, the contents in man being considerable in bulk, semi-fluid, and probably filling the ascending as well as the transverse colon. The large intestine is supplied with nerves from the central nervous system. These run partly in the sympathetic system along the colonic and inferior mesenteric arteries and partly in the pelvic visceral nerves, which are derived from the sacral cord and pass direct to the pelvic viscera. In addition, the large intestine possesses a local nervous system, presenting the same structure as that found in the small intestine. As has been shown by Elliott, the movements of the large intestine differ considerably in various animals, according to the nature of the food and the part played by this portion of the gut in the processes of absorption. In the dog the process of absorption is almost complete at the ileocolic valve, whereas in the herbivora the greater part of the processes of digestion and absorption occurs in the colon and cecum. Man takes an intermediate position as regards his large intestine between these two groups of animals. Here for the first time is met a form of movement which is not found elsewhere in the gastro-intestinal canal, namely, the so-called anti-peristaltic contraction. This does not involve an advancing wave of inhibition and must not be regarded as representing the exact antithesis of a peristaltic wave. The effect of these waves is to force the food down into the cecum, regurgitation into the ileum being prevented partly by the obliquity of the opening, and partly by the tonic contraction of the ileocolic sphincter. The result is a thorough churning up of the contents and its close contact with the intestinal wall. The movements are rendered still more effective by the sacculation of the walls of this part of the large intestine.

A true peristaltic wave takes place in the cecum, driving the food down the intestine into the transverse part. These waves die away before they reach the end of the colon and the food is driven back again by waves of anti-peristalsis. Occasionally more food escapes through the ileocolic sphincter from the ileum so that the whole ascending and transverse colon may be filled with a mass undergoing a constant kneading and mixing process. The result of this process is the absorption of the greater part of the water from the intestinal contents, as well as of any nutrient material, and the drier part of the intestinal mass collects towards the splenic flexure, where it may be separated by transverse waves of constriction from the more fluid parts which are being driven to and fro into the proximal and intermediate portions. By means of true peristaltic waves these hard masses are driven into the distal part of the colon, and the distal

colon must now be regarded as a place for the storage of feces and as the organ of defecation. The storage of waste matter takes place chiefly in the sigmoid flexure, which with the rectum represents the distal portion of the colon. The distinguishing feature of the distal colon is its complete subordination to the spinal centers. It probably remains inactive until an increasing distension excites reflexly through the pelvic visceral nerves a complete evacuation of this portion of the gut. In man, in addition to the stimulation of these nerves, the emptying of the colon is largely assisted by the contractions of the voluntary muscles of the abdominal walls and pelvic floor. This section of the rectum is finally emptied by a forcible contraction of the levator ani and the other perineal muscles, and this contraction also serves to restore the everted mucous membrane. In man, as Hertz has shown by the skiagraphic method, the pelvic colon becomes filled with feces from below upwards, the rectum remaining empty until just before defecation. In individuals whose bowels are opened regularly every morning after breakfast the entry of feces into the rectum gives rise to a sensation of fullness and acts as the call to defecation. If no response be made, the desire to defecate passes away, since the rectum relaxes and the fecal mass no longer exercises pressure on its wall. Hertz has shown that the minimal pressure required to produce the call to defecate varies from 30 to 40 millimeters Hg, according to the length of the gut which is the seat of distension.

ROENTGENOGRAPHIC EXAMINATION.

The bismuth food passes so rapidly through the small intestine that the determination of a peristaltic wave is a matter of great difficulty. With the exception of a small mass in the bulbous duodeni and the collection of masses in the lower portion of the ileum, little is seen definitely in normal cases.

The bismuth meal passes through the duodenum in from 25 to 60 seconds. Special technique is necessary to show successfully the duodenum in normal subjects, departures from the normal being more easily seen. Observations on the duodenum may be made with the patient standing up or lying on the X-ray couch. It is better to examine the patient in the upright position, centering the tube over the pyloric end of the stomach. A slight degree of lateral rotation of the patient throws the duodenum well into the field of vision, and on screening, the bismuth is seen throughout its whole course along the duodenum. There are instances in which a hesitancy of the bismuth

mass at the duodeno-jejunal junction may be observed, but this is rare, because as a rule the bismuth runs over the junction and drops down through the straight jejunum quickly. From this point downward the bismuth spatters generally throughout the coils of the small intestine, there being no very definite arrangement possible. The transit seems to be so rapid that in small and detached plates the coils are not distinguishable. Occasionally, however, plates are obtained which show a fairly regular arrangement of the coils of the small intestine lying mostly in the center of the abdomen to the left, the lower portion of the coils being quite low. Apparently there is some slowing in the lower end of the small intestine, where the bismuth collects in masses, outlining the lower end of the ileum in most instances, particularly when there is some pathological condition about the ileocecal valve region. In the cecum and ascending colon a retardation is met with, and from this outward there is found what is represented as the Cannon ring in the proximal portion of the transverse colon.

Except for a few moments each day the large intestine is quiet. It is not a uniform tube, but sacculated and divided longitudinally into a number of nearly separate compartments, the well-known haustra. This is the reason that its contents remain distributed along the course of the bowel and do not fall to the lowest point. In turn the circular layer of fibers of the large intestine are particularly active when there is content in that portion of the gut. When a peristaltic wave starts at any point in the course of the large intestine, the part in front alters in shape and loses its sacculation. This is effected not by a relaxation of the longitudinal bands, but by a general toning up of the circular fibers of the haustra. Thus the bowel in front of the wave becomes of uniform but diminished caliber. The advancing wave consists of a very firm contraction of the circular wave, each fiber contracting in its turn and remaining strongly constricted for many minutes after the front of the contraction wave has passed on. In this way the column of feces is propelled from behind by an even pressure which advances steadily along the gut as fiber after fiber is included in the peristaltic wave. It is thus forced through a small cylindrical tube, the walls of which have acquired sufficient tone to resist the pressure of the advancing mass. The firm, more or less solid contents form a pliable, cylindrical mass which follows the course of the bowel bending around the flexures. The rear of the moving fecal mass is conical shaped, showing the regular manner in which the tonic contraction passes into firm contraction. There is no possible chance of the feces passing back again for each circular fiber

PLATE I

Plate of a case of gastro-enteric hypermotility, showing a spattering of barium throughout the small intestine so perfect that the coils can be made out in anatomy and position. (X-ray by author.)

PLATE II

Showing mass movement. *A*, Sigmoid. *B. B.*, Trail left after the advance to it. *C*, A little left in the splenic flexure. *D*, Cecum and ascending colon. *E*, Cannon's ring. (X-ray by author.)

remains firmly contracted for some time. Often we may see a thin line of feces remaining in the bowel after the wave has passed, the lumen of the bowel being reduced to a thin thread. After some minutes the continuous thin line of feces begins to break up showing that the circular fibers are relaxing and the lumen of the gut is

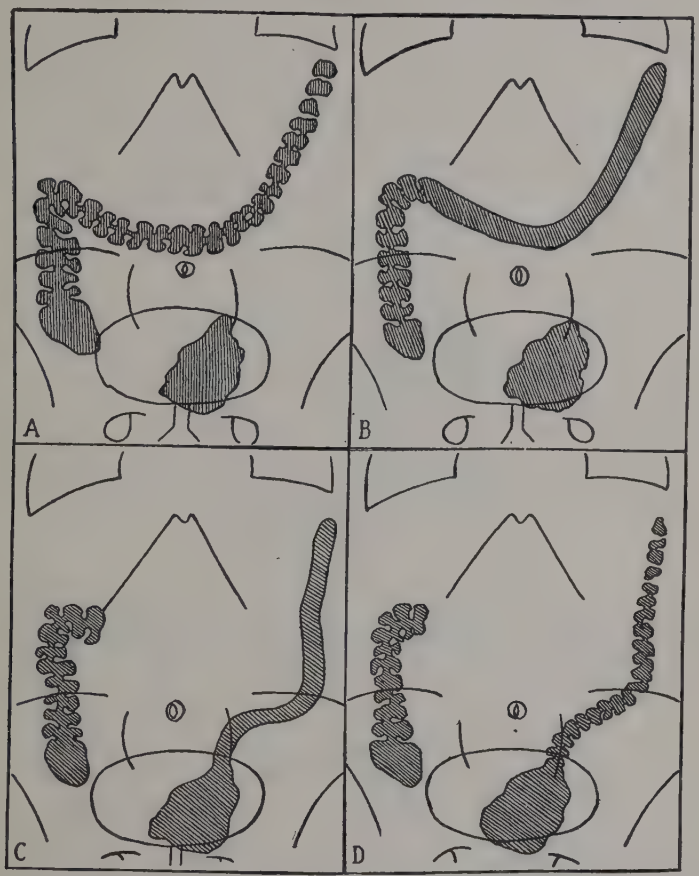

Fig. 11.—Four drawings from Holzknecht's original article illustrating the mass peristaltic movement. In *A*, the colon is uniformly filled from cecum to splenic flexure.

opening up. This is the normal mechanism, although there are irregularities and deviations from the normal type.

In following the bismuth meals through the alimentary tract one is often struck by the fact that the large intestine scarcely ever shows active movement, although its content has advanced considerably since the last observation. It is probable that the principal onward

movement of the large intestine is the mass movement described by Holzknecht. It is a striking phenomenon when seen and consists in the bowel content suddenly losing its haustral markings, and being formed into an ovoid sausage mass with perfectly smooth edges and rounded at both ends. This mass travels at about twice the rate of the peristaltic wave of the stomach, the distance traveled varying with the circumstances. When the mass comes to rest the haustral indentations reappear, quickly if the bowel content is semi-fluid; more slowly if the bowel content is firmer. It is estimated that these mass movements occur about six times daily. It is assumed that the respiratory movements and the filling and emptying of the

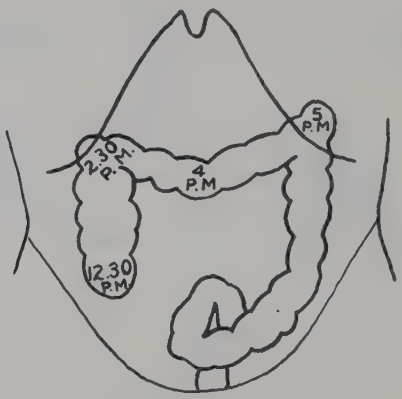

Fig. 12.—X-ray observation of normal colon in man and position attained by its contents at different periods after a meal containing bismuth. The bismuth meal was taken at 8 A.M. Times of arrival at different levels marked in colon. (*Hertz.*)

stomach are important factors in the shifting of the bowel content. It has been observed by the author, however, that active defecation itself is sometimes followed by a mass movement, particularly when the entire sigmoid has been emptied.

It seems quite impossible by palpation or manipulation to shift the content of one portion of the colon proximalward and the same may be said of moving it onward. Unless it is fluid, it does not seem possible with any degree of assurance, to move the content of the ascending colon onward into the transverse. Nor is it possible to push the content of the proximal transverse back into the ascending. It is probable that a status of tone keeps the content at the position it occupies. It may be said that the haustral churning is also met with in the small intestine, although there the actual radiographic evidence is nowhere near as perfect as in the large.

According to Kant, whose finding is now generally accepted as fact, peristaltic waves in the stomach originate in a tonic constriction ring, the exact location of this ring varying with the tone and degree of filling of the stomach. A similar tonic constriction ring exists in the transverse colon (Cannon's ring). From this ring there travel anti-peristaltic waves toward the cecum. The exact location of this varies with the tonicity and the degree of distension of the proximal colon. Peristaltic waves travel distally from this ring onward in the transverse, the descending colon and the rectum. The entire peristaltic waves of the ascending colon are somewhat livelier than the peristaltic waves distal to the Cannon ring. By means of the X-ray this tonic constriction ring in mass is easily observed. Thus it may be said that in the stomach a bismuth or barium meal should pass out in within four and a half hours, and that the head of the barium column should have reached the cecum by that time. The barium meal should have passed into the colon by the eighth hour, or, at most, the tenth, when the head of the column should have reached the middle of the transverse colon. The ascending colon should be reached in from nine to sixteen hours, and the colon should be practically empty of barium at the thirty-sixth hour, no purgatives having been taken during the time of the observation.

CHAPTER IV.

Anamnesis.

THE interrogation of the patient, the ability of the history taker to discern and marshal salient points and reject those which are superfluous, is of the greatest value in gastro-intestinal conditions. It may honestly be said that in practically half of all the instances of gastro-intestinal affections a diagnosis is made, or strongly suggested, from the history alone, and that much that is done in the way of investigation is confirmatory. Of course there are numbers of cases wherein one must work assiduously to accomplish a diagnosis. But yet it is often common enough to deduct from the history alone a suggestive diagnosis which is clinically well worth the while. Mayo has stated that history comprises 50 per cent. of the diagnosis, the X-ray about 40, and the laboratory 10. I agree perfectly with this in so far as history is concerned, but I do not agree in so far as the X-ray and the laboratory are concerned. Thus it will be seen that the taking of a history is a very important part of the work, one that should not be relegated to an assistant or to a subordinate. My experience has been that history-taking is seriously neglected by the medical men now in practice, and it represents much of the value of the older type of physicians that they spent much time in taking careful history. All that has already been said in my text book on the diseases of the stomach, holds good in so far as the intestines are concerned, and there will be only included here such things as deal more particularly with enteric conditions.

There is the important group of symptoms connected with chronic excessive intestinal toxemia that deserves the first attention. Emotional irritability and inclination to mental depression; an early onset of muscular and mental fatigue; gradually developing anemia which is difficult to benefit by means of the administration of iron; the steady downward tendency year after year, the patients always being short of robust, and less capable of rapid recuperation even under most favorable hygienic conditions; the indications of premature senility, and atrophic processes, not limited to fat, muscular and blood tissues alone; those which come from various pathological conditions in the enteric tract, such as gas, meteorism, more or less indefinite types of distress; constipation or diarrhea; recurring neuralgia; persistent forms of eczema, and various other types of dermatological conditions; various functional disturbances in the eye, such

as specks and floating objects, dimness of vision, etc.; inability to maintain one's standard of work, losing endurance before the day is over and from insufficient amount of physical or mental labor. These and many others, have to do in significance with the diagnosis of a chronic intestinal toxemia.

Then there is the matter of the catarrhal conditions from duodenitis to sigmoiditis in connection with the local distress in the abdomen, manifestations of mucus in the stools, varying states of affairs in regard to regular movement of the bowels, etc. There is the pain which accompanies intestinal ulceration of various types, particularly the diarrhea which comes when an ulcerative process is well advanced and in the large intestine, or down in the rectum; and there is always to be remembered the distress attendant on the infective conditions such as typhoid, etc. There is the history in botulism—that is, the history of cause; the chronic history of dysentery in connection with that diagnosis, the important significance of the stools, numbers each day, etc. Then comes the matter of appendicitis, as to whether the patient had attacks of pain in the lower right abdominal fossa before they came to the physician for diagnosis; the steady decline and evidences of dotage in cases of arteriosclerosis; the history of pain, usually on the left side in colon diverticular conditions, particularly in connection with abscess, wherein of course, the matter of fever, leucocytosis, and clinical findings would be important; the history of relief from distress in the abdomen in cases of enteroptosis, such as the relief experienced when prone as compared to when up and about. The history of constipation in connection with the diagnosis of malignant disease of the intestines—that is, initial constipation finally followed by more or less movements of the bowel with the presence of blood, mucus, pus, etc.; the change of routine in life as causative factor in connection with the symptom of constipation; establishment of diarrheal conditions from neurological disturbances; constipation accompanying plumbism; the subject of hemorrhage from the bowel and intestinal obstruction; the history of the acute onset of pain in connection with various stone formations, whether in the pancreas, gall-bladder, etc.; history of the enlargement of the abdomen, such as is found in cases of accumulation and of new growths in the abdomen, and finally, all the points which have to do with diseases of the rectum and anus.

Then comes the importance of symptoms, as to how they have progressed and their character, and changes; matters of loss of weight; all that pertains to headaches, vertigo, changes in appetite, thirst, pain, distress, abnormal sensations of various kinds, such as bloating, full-

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ness, weight and pressure, belching, regurgitation, pyrosis, pains in various parts of the abdomen, vomiting, character of stools, general health and strength. These and many others are all important in connection with the anamnesis in this type of work.

HISTORY RECORDS.

The advantages of keeping records of the cases examined, the treatment prescribed and results from same, are of inestimable value, and the busier the physician the more necessary it is that such details pertaining to a case be noted in writing and not trusted to memory alone.

I have seen no reason for changing the plan of history sheets mentioned in my text-book on the stomach. These history sheets, now used in numberless cases, have proven to be most satisfactory, both in the office and clinics. Inexpensive, handy, easily filed, quickly gotten, and read within a minute or two after being spread before you.

Name _____ Address _____ Diagnosis _____

Date _____ 191 S. M. W. D. Children Occupation _____ Age _____

Family History _____

Personal History _____

Present Illness _____

EXAMINATION

Fig. 13.—Author's examination blank. Printed in the form of a 3-ply folder. Size when spread out 8 by 15 inches. Above shows arrangement of printing. Pages 1 and 2.

TEST MEAL

Date ..... No. .....
Simple or Mixed .....
Time removed .....
Amount .....
Reaction { Congo Red ..... / Litmus ..... }
Free Hcl. { Phloroglucin ..... / Resorcin ..... / Amido-Benzol ..... }
Töpfers { Free Hcl. ..... / Total Acidity ..... / Combined Hcl. ..... / Total Hcl. ..... }
Lactic Acid { Uffelmann ..... / Kelling ..... }
Ether .....
Pepsin .....
Starch { Erythrodextrin ..... / Achrodextrin ..... / Maltose ..... }
Motor tests .....Gas result .....
Bile .....Mucus .....
Blood .....Pus .....
.....

GROSS AND MICROSCOPICAL FINDINGS

.....
.....
.....
.....
.....
.....
.....
.....
.....

FECES

Date ..... No .....
Color .....
Consistency .....
Surface Ex. { Blood ..... / Pus ..... / Mucus ..... / Bacteria ..... / Tissue ..... }
Reaction .....
Food Particles .....
.....

BACTERIOLOGICAL

Gas results (water and feces).....
Reaction of gases .....
H_2S .....
Reaction of fluids .....
Gas results (dextrose bouillon) ..
Per cent. of Bacteria .....
Gram—P. Bac. ...Gram—N. Bac...

CHEMICAL TESTS

Putrefaction .....
Blood (Occult) .....
.....
.....

REMARKS

.....
.....
.....
.....
.....
.....
.....
.....
.....

URINE

Date ..... No .....
Specific Gravity .....
Quantity, 24 hrs. .....
Reaction .....
Appearance .....
Color .....
Indican .....
Other Chromogens .....
Bile .....
Blood .....
Pus .....
Acetone .....
Diacetic Acid .....
.....

QUANTITATIVE

Urea .....
Albumin .....
Glucose .....
Oxalic Acid .....
Uric Acid .....
Nitrogen, total gms. .....
Ammonia, total gms....Amt. N....
.....% of total N.
Sulphates .....
Phosphates .....
Chlorides .....

MICROSCOPICAL

Mucus .....Blood .....
Pus .....Casts .....
Phosphates .....Urates .....
Calcium Oxalate ...Epithelia ...
.....
.....
.....

BLOOD

Date ..... No. .....
Hemoglobin .....
Red Cells.....in a Cubic Mm.
Color Index .....
Erythrocytes .....
Size .....
Shape .....
Degeneration .....
Erythroblasts .....
White Cor. .....in a Cubic Mm.
Differential Count, 1..1 { Polymorphonuclear .....% / Large Mononuclear ....% / Lymphocytes .....% / Mast Cells .....% / Eosinophiles .....% / Myleocytes .....% }
Parasites .....
.....
.....

SPUTUM

Date ..... No. .....
Tub. Bac. .....
Pneu. Coc. .....
Influen. .....
Pus Organ .....
Fibers .....
Pus .....
Blood .....
Crystals .....
Casts .....
Spirals .....
Parasites .....
.....
.....
.....

Dr. A. Bassler's Case Record Blank.

Author's examination blank. Arrangement of printing on page 3.

Fig. 14.—Upper part of the reverse side of author's examination blank, for observation notes and treatment.

FRACTIONAL TEST MEAL

140													
130													
120													
110													
100													
90													
80													
70													
60													
50													
40													
30													
20													
10													
0													
Time	¼	½	¾	1	1¼	1½	1¾	2	2¼	2½	2¾	3	3½
Blood													
Bile													
Mucus													
Lact. Ac.													
L. A. Bac													
Residue													

Blood { Culture / Wasserman / Fixation }

BLOOD CHEMISTRY

	12-15	Mg.
Urea N.		
Sugar	.09-.12	P. C.
CO_2. C. P.	50-75	C. C.
Creatinine	1-2	Mg.
Uric Acid	2-3	Mg.
Non-Pro. N.	25-30	Mg.
Cholesterol	.14-.17	P. C.
Chlorides (NaCl)	.45-.50	P. C.
Diastatic Activity	15-20	—

SPINAL FLUID

REMARKS

Separate laboratory slip which fits in the folder.

CHAPTER V.

The Examination of Patients.

GENERAL INSPECTION.

In a broad way, inspection of the body takes into consideration every external symptom in any way connected with the diagnosis of internal diseases and it comprises all matters that are connected with diagnosis in general, because many abdominal cases show disease elsewhere in the body and it is not uncommon to meet with

Fig. 15.—Examining table used by the author.

cases having symptoms in the abdomen while the disease itself is situated outside of the abdominal cavity. This being a wide field, it is not necessary to deal with it extensively here. I will confine myself to abdominal diagnosis alone.

The assistance of daylight is more advantageous for estimating such details as color of the skin and general illumination of the patient. Artificial light is better in obtaining details and shadow effects and for the concentration of a flood light upon a certain part of the abdomen while all the rest is dark. I have had no reason to change my good opinion of the examining table which I use—a table 6 feet long, and 28 inches wide, and supported by four strong posts 30 inches from the floor, the top of which table is covered by a thin mattress of black oilcloth.

The examination is begun with a general inspection of the body; that is, all that pertains to the condition of the hair, the pupillary

reflexes, the condition of the mouth, the color of the skin and general appearance of the body as a whole. This should be followed by noting the reflexes, both those of the elbow and forearm, as well as the patellar reflexes; the general examination of the heart and lungs; the noting of the condition of glands particularly enlarged ones, such as those connected with malignant disease and the epitrochlear gland in its significance in syphilis. The odor of the breath may be of considerable value in diagnosis. Inspection of the abdomen may give details of diagnostic value, although in other cases again, no useful information may be gained. As a routine I now perform it at the

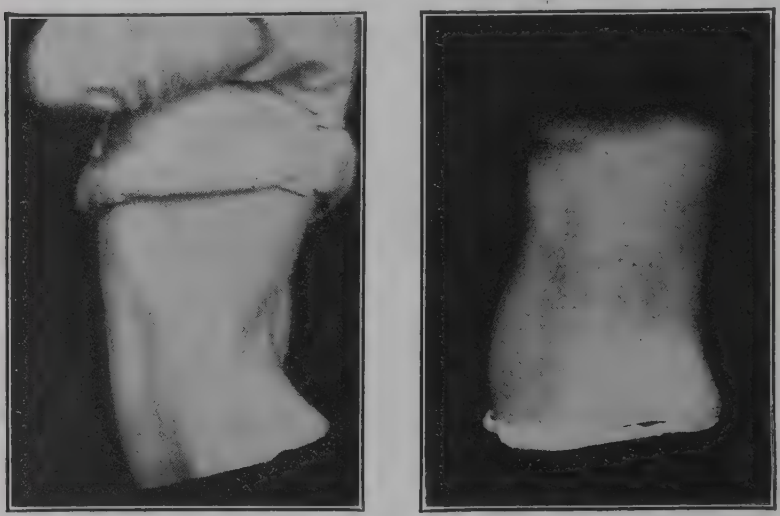


Fig. 16.—Two types of normal abdomen in women who have borne children. (The first is a decidedly better type than the second.)

end of a general physical examination. It is important to note the nutrition of the walls, shape, size, bulging (when present whether local or general), excursion or retraction of the abdomen as a whole during respiration, and also whether peristaltic movements of the gastro-enteron are visible.

The appearance of the skin of the abdomen is of value in the following ways: Many linear albicantes are evidence of past pregnancy or marked reduction in weight, both of which conditions may predispose to ptosed states of the abdominal organs. Eruptions of cutaneous diseases, and the rose spots of early typhoid may be seen. Some knowledge of the gross appearance of the common forms of skin diseases is of value, particularly in being able to differentiate irritative rashes due to toxic absorption from the colon and

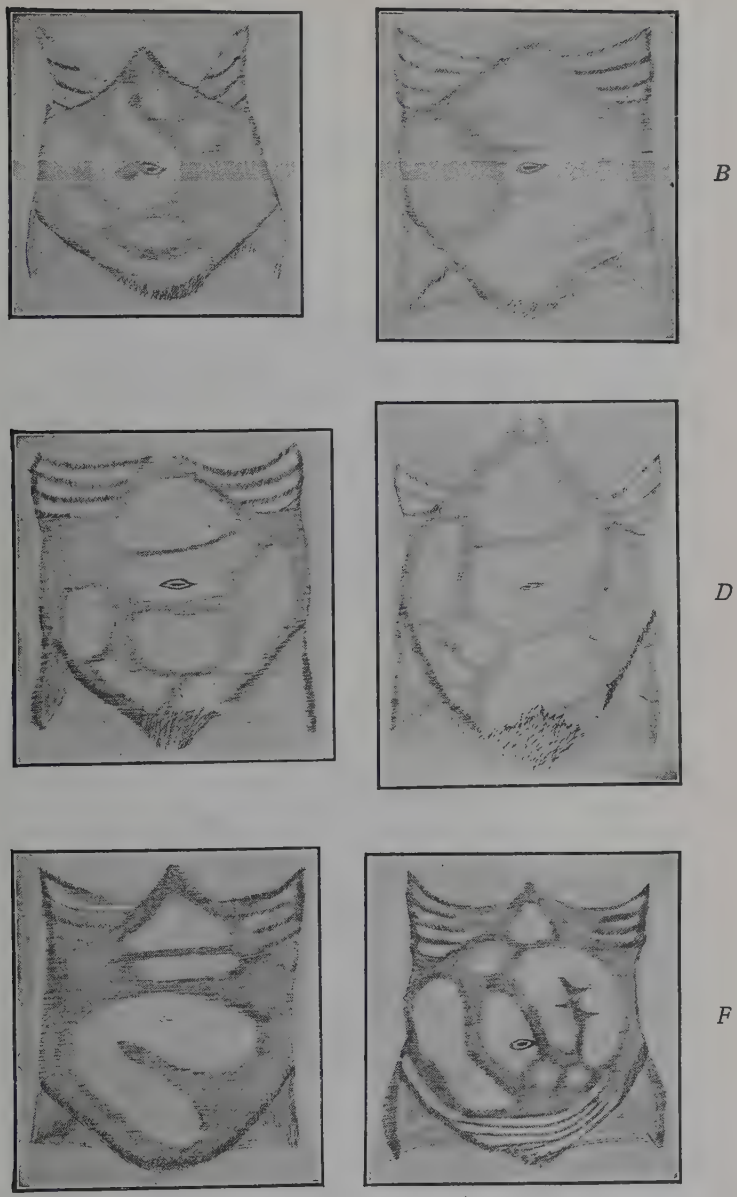
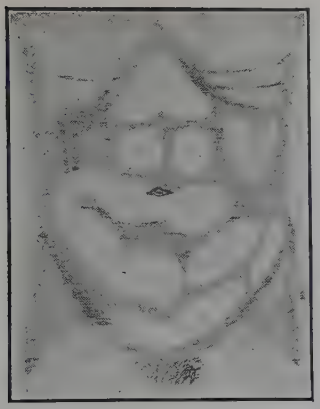


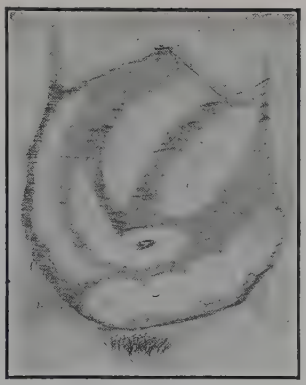
Fig. 17.—Visible prominences of the abdomen. (After *Nothnagel.*) *A,* Carcinoma of hepatic flexure of the colon, ileus, peristalsis in the right colon and small intestine. *B,* Stenosis of ascending colon in region of hepatic flexure. *C,* Stenosis of sigmoid flexure (following dysentery). *D,* Chronic ileocecal valve invagination. *E,* Stenosis from cicatrix in lower part of sigmoid flexure. *F,* Stenosis (cicatrices following tuberculous ulcer) in ascending colon at hepatic flexure. Peristalsis in ascending colon and small intestine.

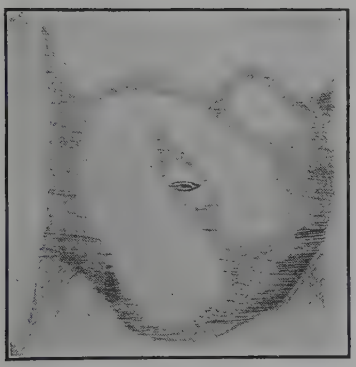
the excretion of these poisons by way of the skin from the more chronic types of skin disorders. The yellow tint of jaundice or leukemia, or the fainter yellow of toxemic jaundice; the very light,

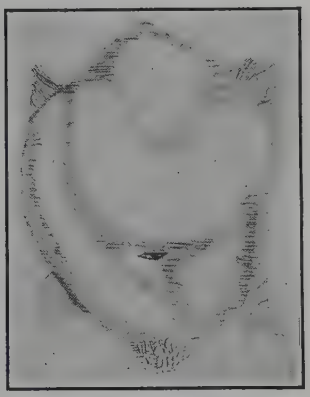
Fig. 18.—Visible peristaltic movement in carcinoma of the pylorus. *A*, Normal condition of the epigastrium with slight sulcus from the ensiform to the umbilicus. *B*, Bulging of the epigastrium due to peristaltic movements.

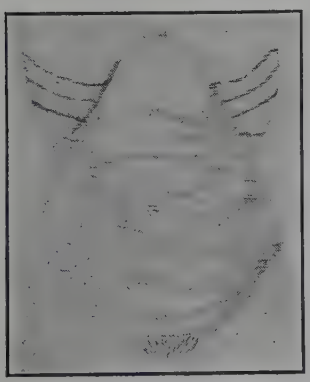
yellowish brown of malignant cachexia, and the faint brown of hepatic cirrhosis may be observed. A general brown or browning-black color is seen in Addison's disease, although here the pigmentation may be very faint and only present on the upper extremities.











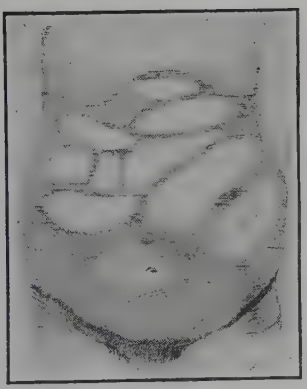


Fig. 19.—*A*, Stenosis of the lower ileum due to peritoneal adhesions. *B*, Stenosis of the sigmoid flexure at left ileopectineal line. *C*, Stenosis due to carcinoma at lower part of sigmoid flexure. *D*, Cicatricial stricture in region of splenic flexure of colon. *E*, Vigorous peristalsis of small intestine (koprostasis). *F*, Stenosis of sigmoid flexure and peristalsis of large and small intestine.

General enlargement of the superficial abdominal veins may be seen in greatly dilated stomachs, cirrhosis or tumor of the liver, and obstructions of the vena cava. The so-called caput medusæ may be seen in obstruction of the portal circulation or hepatic cirrhosis. Note should be made of absent or limited abdominal respiratory movement (peritonitis), and excessive abdominal breathing (diseases of the lungs).

To note the nutrition of the wall is of much value in diagnosing gastro-intestinal cases. In malignant disease the subcutaneous fat of the abdomen is more or less lost. Likewise is this true in atrophic states of the glandularis of the gastro-enteron, cases of atony or dilatations in various parts of the viscera. The shape, size and bulg-

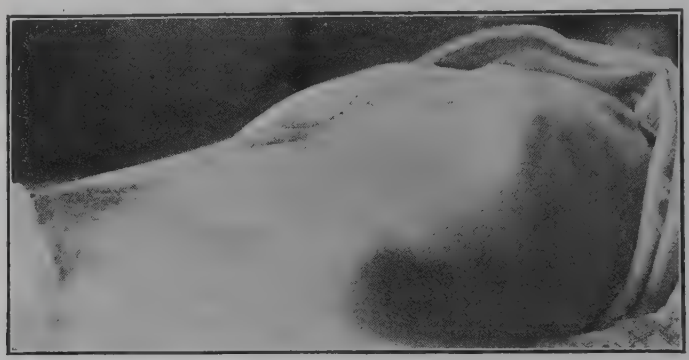


Fig. 20.—Enlargement of the liver due to congestion and caused by endocarditis. No other abdominal symptom.

ing of the abdomen often give important diagnostic suggestions. The abdomen which is large in size, pendulous in the upright position and which flattens in the recumbent position, means relaxed abdominal walls, a common result following pregnancy, and is often seen in cases of more or less splanchnoptosia and chronic colitis. Note should be made of the shape and breadth of the body, for the long thorax-abdomen type of individual often has prolapse of the abdominal viscera and endocrine disturbance; the same may be said of those individuals who have a widely flaring ilia and a wide costal arch. Under favorable conditions in thin-walled subjects, the outline or bulge of the stomach or colon may be seen. I have seen the entire right side of the colon project the abdomen prominently forward. Furthermore, the abdominal wall may be raised and present a local bulging from the mass of a neoplasm connected with any of the abdominal organs—even the kidneys which are deeply situated.

The opportunity is frequently offered to observe the peristaltic waves of the stomach and the vermicular movements of the intestines. They present themselves as a series of rolling, rounding ele-

Fig. 21.—Dorsal method of displacement of fluid in an ascitic abdomen. *A*, Upper limit of fluid when lying on the back. *B*, Upper limit of fluid when slightly raised.

vations which increase and subside. In the stomach they usually run from left to right; in the intestines they are liable to run in any direction, commonly accompanied in stenosis of the intestines by a squirting sound as the fluid passes through the stricture. Instances

are observed of obstruction in the region of the ileocecal valve when the coils of small intestine may lie one above the other in the center of the abdomen. If the stenosis is in the lower colon, or high in the rectum, the distention is around the confined limits of the abdomen; corresponding to the location of the colon. Should the distention and peristaltic movements be confined to the upper and left quadrant

Fig. 22.—Ascites due to interference with returned circulation in the pelvic veins from pelvic phlebitis following parturition. (New York Polyclinic Hospital.)

and running toward the pylorus, a stenosis in that region is suggested. Note should be made as to the quantity of fat in the abdominal wall, particularly in connection with the diagnosis of obesity, when the abdomen is uniformly arched, lying or standing, and perhaps there are small bulgings somewhat lobulated owing to the arrangement of fat. The navel is depressed in obesity because the connective tissues thereabout are so rigid that fat cannot be deposited. The attempt to grasp the outermost tissue between the fingers will solve any doubt.

Meteorism (tympanites) may cause abdominal distention. It then lessens or obliterates the umbilical depression unless the wall is thickened by edema or fat. The contour of the stomach or of the intestinal coils can frequently be seen through a thin abdominal wall, especially when peristalsis is active, or when the organs are dilated, particularly if the dilatation be dependent upon the stenotic function

Fig. 23.—Abdomen in a clinic case of splanchnoptosia showing the convexity above and protuberance below in the lower zones of the abdomen. The sag of the abdomen produced the side relaxation shown by the bulging over Poupart's ligament.

in the digestive tract. When there is pyloric stenosis (ileus, intestinal tumors), by noting through the abdominal wall the contour of a specially distended and perhaps completely immobile coil of the intestines, the position of the obstruction can sometimes be determined. The paralytic immobility and the absence of intestinal murmurs on auscultation are important signs in the diagnosis of acute, diffuse peritonitis.

Free accumulation of air in the peritoneal cavity from perforation of the stomach or intestines is characterized by uniform distention of the abdomen without visible stomach or intestinal contour.

Collection of fluid also produces an apparently uniform distention of the abdomen. In the dorsal decubitus the fluid when under no great tension is mostly accumulated in the lateral portion, so that the abdomen seems to be disproportionately broad. In case the fluid reaches as high as the navel, this slightly protrudes. Percussion of an abdomen containing free fluid produces a dullness of the

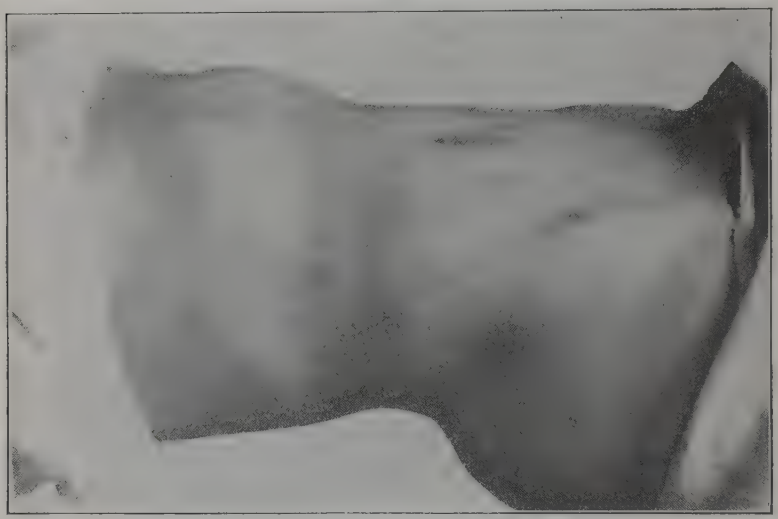


Fig. 24.—Same as Fig. 23. Dorsal position. Shows the characteristic flattening of the abdomen due to the recession of the ptosed internal organs, and the bulging of the flanks.

dependent portions. Still more essential to the diagnosis of free fluid is the so-called fluctuation wave which can sometimes, when the patient is moved, be appreciated.

The presence of distended veins of the abdominal wall aids the causal diagnosis of free fluid in the abdominal cavity. It may be limited to the sides of the abdominal walls, and if the examination shows that the veins conduct the blood from below upward they present the type of collateral circulation observed most plainly in a thrombosis of the inferior vena cava. In this case a part of the blood which should proceed through the vena cava inferior is deflected and flows directly from the lower extremities through the veins of the anterior abdominal wall into the vena cava superior. Any fluid effusion in the

abdominal cavity may compress the inferior vena cava enough to cause some obstruction, hence this sign of a collateral circulation is of slight value unless the venous distention be very marked, when

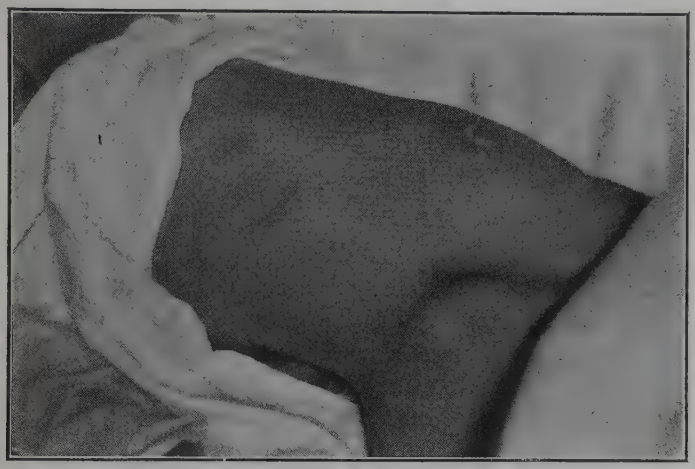


Fig. 25.—Prominence of the abdomen due to enlarged spleen in a case of myelogenous leukemia.

it would point to an actual thrombosis of the inferior vena cava. On the contrary, distended veins which occupy the umbilical region would point to an anatomic obstruction of the portal circulation,

Fig. 26.—Tumor of liver displaying marked prominence.

such as is met with in cirrhosis of the liver and thrombosis of the portal vein.

Large ovarian tumors or other abdominal cysts are differentiated from effusion of free fluid by inspection, palpation or percussion.

With them the greatest degree of prominence and resistance is in the median line of the abdomen, not in the more dependent portions. It is usually necessary for all the other methods of examination to be employed for a certain differentiation.

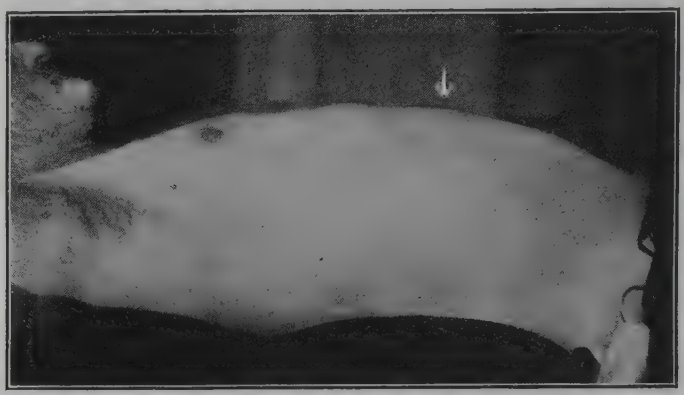


Fig. 27.—Gastric carcinoma, arrow pointing to prominence of epigastrium caused by the growth.

Enteroptosis following pregnancy presents on inspection a peculiarly characteristic and practically clinical picture. The condition depends upon the relaxation of the abdominal walls and of the mesen-

Fig. 28.—Showing a moderate amount of abdominal distention due to perityphlitic adhesions following a suppurative appendicitis for which operation was performed five months before.

tery. It is also most frequently observed in the female sex and is due either to a marked loss of sub-peritoneal, sub-mesentery or subcutaneous fat and to an atrophy of the abdominal wall muscles, or

an impaired tension of the stretch of the abdominal walls of the peritoneum. It is almost always associated in extreme cases with a loss of fat in the abdominal wall and of the abdominal content, a certain degree of atrophy and frequently even a spreading of the recti muscles. As a consequence the abdominal organs are abnormal and mobile, the liver and kidneys in the erect position are situated lower than normal, the right kidney especially gradually draws down a mesentery for itself and becomes a movable, even a genuine floating kidney. In the

Fig. 29.—Prominence of abdomen due to enlargement of spleen in myelogenous leukemia. (New York Polyclinic Hospital.)

erect position in these people a veritable pouch is noted, and through the gaping breach between the recti muscles a considerable portion of the abdominal content sometimes projects like a hernia. Enteroptosis can exist in the virgin female and in the male, the organs being very prolapsed, and no change in the abdomen contour noted. These are mainly in the congenital and endocrinopathic cases.

Empty intestines in inanition, that is, from starvation, esophageal stenosis, or in cases of malignant disease produce a very decided retraction of the abdomen. A similar appearance is observed in tubercular meningitis. The so-called scaphoid belly is due to a contraction of the intestinal muscles, and perhaps abdominal muscles as well. Inspection sometimes reveals local prominences due to cysts, tumors,

enlargement of the abdominal organs and their mobility with respiration.

The general types of abdomens may be classified as follows:

Obese abdomen	Ascitic abdomen
Scaphoid abdomen	Gaseous abdomen
Abdomen of pregnancy	Enteroptotic abdomen

In the matter of local abdominal enlargements the following may be mentioned: Enlargements of the upper left side, such as are observed in enlargements of the spleen, hepatic tumors of Banti's

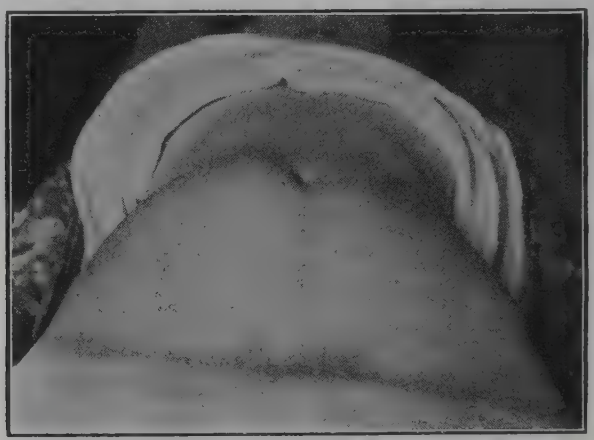


Fig. 30.—Tuberculosis of right kidney.

disease may bulge the abdomen on that side to the umbilicus or perhaps as low as the iliac fossa and pelvis. Enlargements of the upper abdomen below the right costal arch are suggestive of lesions of the liver and gall-bladder, congestion, cirrhosis, amyloid disease, malignant and gummatous tumors, echinococcus cyst, abscess of the liver, empyema, or cancer of the gall-bladder. Or a swelling here may be due to a fecal or malignant tumor of the ascending colon in the neighborhood of the hepatic flexure. Right-sided subphrenic pyopneumothorax may produce an immobile mass in the hepatic region and perhaps in the epigastric.

Enlargements of the epigastrium proper are referable chiefly to a distended or dilated stomach, neoplasms of the pylorus, left lobe of the liver, pancreas, transverse colon, and omentum. Aneurism of the abdominal aorta is recognized as a pulsatile tumor in the median line of the epigastrium. I have observed large gall-bladders slightly to the

right of the median line of the epigastrium; to the left, immediately above the colon an effusion into the lesser peritoneal sac.

Local enlargements in either flank commonly depend upon lesions of the uterus and adnexa—pyosalpinx, ovarian, uterine and ligamentous tumors; perhaps an ectopic gestation; or a mass in one of the

Fig. 31.—Suggestion of liver enlargement seen in the bulging of the mid-portion of the abdomen. (German Poliklinik.)

iliac areas may mean tuberculosis or malignant disease of the peritoneum. A psoas abscess may bulge, fluctuate and point in the groin, either below or above Poupart's ligament, while an iliac abscess commonly appears above the outer end of this landmark. In the right flank are found palpable tumors of appendicitis, or mass adhesions and in this region may also be found the tumors due to neoplasms of the cecum or ascending colon and to fecal impaction in these portions of the intestines. In the left flank similar obstructive lesions of the

descending colon and sigmoid flexure are possible causes of a local swelling, additional factors to be borne in mind being splenomegaly and splenoptosis, and peridiverticular abscesses.

In the region of the umbilicus, a distention is suggestive of gastric dilatation and displacement; less commonly of enteroptosis or of other visceral ptoses—a displaced spleen or kidney may be more conspicuous near the navel than it is in the epigastrium or in the flanks. The umbilical region is also the site of hernia and of tumors caused by tuberculous peritonitis, and by neoplasms of the stomach, gut, omentum and mesentery.

Distention of the lower abdomen above the pubes, if not obviously a sign of pregnancy, may mean an overdistended bladder, or, very rarely pyo or hematometra. Additional factors of hypogastric swellings include the diseases of the female genital organs, the appendix, and the peritoneum, as well as inguinal hernia.

On inspection massive abdominal tumors may account for an apparent general enlargement of the abdomen; especially is this true of the ovarian cyst, the pregnant or fibroid uterus, the leukemic spleen, an overfull bladder, and the cancerous gut and peritoneum which at first glance simulate a universal abdominal distention.

PALPATION OF THE ABDOMEN.

The first requisite for palpation of the abdomen is the relaxation of the abdominal wall. This is accomplished by the patients lying perfectly prone with their hands to the side, or with their knees raised and somewhat separated. Slow, regular breathing should be advised, and with apprehensive individuals conversation should be engaged in so as to take their mind away from the physical examination that is being conducted. With some individuals it is advantageous to raise the buttocks by putting a number of pillows under them, or to place the patient on the examining table in the Trendelenberg position, which frequently relaxes the abdominal walls to a marked degree, pushing the abdominal organs toward the diaphragm, thus allowing the lower part of the abdomen to be more easily palpated. In conditions of extreme degree of meteorism the giving of an enema to empty the colon of feces and gas is usually worth while. With most individuals palpation may be conducted in a satisfactory way. However, in conditions of extreme tension and extreme sensitiveness it may be necessary to employ anesthesia or to examine the abdomen under conditions of a prolonged hot bath—that is, while the patient is still in the water. Few people enjoy the placing of cold

finger tips upon the abdomen. It commonly excites a reflex spasm in the abdominal walls and makes palpation unsatisfactory. Thus, if the hands be cold they should be warmed. Abdominal palpation should be engaged in with the flat of the hand, allowing the fingers and finger tips to dance up and down as the hands advance around the abdomen. It is my custom to divide abdominal palpation into two parts—namely, superficial and deep. The superficial palpation is conducted by a general swinging about of the hands encompassing the entire abdominal confines, feeling particularly for tender areas on the skin, for the condition of the abdominal wall, and the presence of any localized areas of rigidity such as is observed in cases of ulcer, appendicitis, inflammatory conditions of the peritoneum, etc. This is followed by a heavier palpation wherein effort is made to sink the hands as deeply into the abdominal cavity as is comfortable to the individual. In thin people the spinal column almost to the promontory of the sacrum can be felt.

In palpating painful affections it is a good rule to begin with the painless part, thus gaining the patient's confidence and distracting his attention. Frequently bimanual palpation furnishes very useful results. One hand placed upon the lateral or anterior surface of the abdomen pressing the parts against the other hand in the flanks, perhaps in the vagina or in the rectum.

In all difficult cases it is of decided advantage to palpate in different positions of the body—that is the dorsal decubitus, the right and left lateral, erect, knee-chest position, the elevated pubic posture, and finally the Trendelenberg.

Rubbing the skin of the abdomen with oil, or what I usually employ, talcum powder, frequently facilitates an examination. Jerky or interrupted palpation is often advantageous for certain purposes, wherein the hands or tips of the fingers are placed firmly upon the abdomen and quite powerful but short strokes are employed, agitating the tissues beneath. This method may demonstrate a collection of food in the abdomen or the presence of deeply placed solid masses covered by a fluid which ordinary palpation would not reach. "Dipping" can be recommended to demonstrate the enlargement of the liver or the spleen, benign tumors or distended gall-bladder, and for palpating an abdomen distended with fluid when the ordinary palpation as well as percussion leaves one in doubt.

Abdominal palpation should appreciate the general condition in the belly, both of the walls and abdominal content, palpable boundaries of the organs, their sensitiveness to pressure, and any local resistance and tumors. It therefore is a necessary sequence to inspection.

"Splashing" is a valuable method of demonstrating the presence of fluid and gas in the viscera. Fluctuation in the ordinary sense of the word is not practical in the belly.

Enteroptosis presents to palpation an abnormal slimness and flabbiness in the abdominal wall and hyperesthesia usually in the epigastrium. Often the organs may be palpated and bounded just as if there were no covering. Sometimes a stomach distended with gas is much more readily outlined by palpation than by percussion; on account of its firm resistance like that of an air cushion. Contracted intestinal loops feel like tubes, varying in size from a little finger to a thumb. The haustra of the large intestine can sometimes be plainly felt.

In palpating tumors or pathological resistances we should carefully heed their position, form, size, possibility of outlining their boundary, their consistence, character of their surface, and their sensitiveness to pressure. We should determine, if possible, from what organ the tumor arises, whether it is covered by the stomach or by the intestines, etc. Artificial distention of the stomach or colon will sometimes clear up this part of the examination.

In determining tenderness, very gentle palpation is first employed. If this produces no pain, more force is used. We should then determine whether there is sensitiveness to quiet and regular pressure, or only to displacement of parts, for in many cases the tenderness is produced only by a blow, not by mere pressure, as is frequently the case in peritonitis. After demonstrating the existence of tenderness an attempt should be made to differentiate at what depth this is situated. If the tenderness is more deeply situated, direct pressure posteriorly may be required. In this connection it may be important to mention the superficial tenderness met with in cases of pseudoperityphlitis, a condition of the ileocecal region which often arises, especially since public attention has been more or less attracted to the vermiform appendix.

Deep-seated tenderness and often spontaneous pain are common symptoms of hepatic congestion and of various inflammatory processes, such as, for example, perihepatitis, diffuse hepatitis, acute yellow atrophy, Hanot's cirrhosis, and more especially localized, those due to various pathologies of the gall-bladder and appendix.

Pulsations of the liver which may be caused by the thrust of a hypertrophied heart are to be distinguished from those due to congestion of the hepatic venous channels. In the former the "jogging" character of the impulse, the physical signs of right ventricular hypertrophy, and the absence of tricuspid leakage are the cardinal diag-

nostic points; in the latter the expansile, resilient impulse, the murmur of tricuspid insufficiency, and pulsation of the jugulars form a characteristic trinity of signs. The friction-rub of perihepatitis and the thrill by palpatory percussion over hydatid cysts may be elicited.

With complete abdominal relaxation, enlargement and descent of the liver are readily determined by palpating the upper abdominal zone, with the finger-tips pointing upward toward the hepatic area, or by the bimanual method. Except in the young child, and perhaps in the wasted adult, it is always possible for me to feel the lower portion of the liver, perhaps not its direct edge, because I believe when the edge is palpable, the liver is pathological. But by general

Fig. 32.—Ascites due to decomposition of the heart, caused by mitral regurgitation. In the case the apex beat was at the axillary line and there was congestion of the lungs as well as the general venous system. Cyanosis, dyspnea and a pulsating liver were prominent. (Peoples Hospital.)

dancing of the tips of the fingers in the region of the left lobe of the liver it is possible to make out practically within a quarter of an inch its exact lower edge and its fissure.

In the presence of ascites or of meteorism it may be impossible to feel the lower edge of the liver although it extends far below the margin of the ribs. In the former condition, "dipping" may be tried or aspiration may be necessary to drain off the interposed fluid; in the latter the liver may sometimes be felt by Glenard's method—that is, a strong pressure with the fingers of the left hand upon the right lumbar region, and palpating with the left thumb below the right costal margin, meanwhile exerting deep rotary pressure with the right hand across the upper abdomen, the lumbar pressure forcing the liver down and the abdominal pressure pushing the intestine

away from the belly wall and up against the liver so that it may be felt by the examiner's thumb.

The consistence and contour of the liver may vary greatly in different pathological conditions. In amyloid disease the texture of the liver feels dense and unyielding, its surface being uniformly smooth and even, and its lower border blunt and rounded. Increased hardness of the liver may also result from cirrhosis, cancer, syphilis or capsulitis, and leukemic infiltration. A local resilient, perhaps fluctuating, area elevated above the surface of the organ, suggests circumscribed abscess, hydatid cyst, or gumma.

The contour of the liver is regular and the surface unroughened in the amyloid, fatty, cyanotic, and leukemic conditions, in diffuse non-purulent hepatitis and in generalized abscess, in the enlargements associated with febrile states, biliary obstruction, pseudoleukemic anemia, Banti's disease, and other primary anemias. In Hanot's cirrhosis the surface of the liver is generally smooth but might be a little roughened. It is rough, nodular, or lobulated in atrophic and syphilitic cirrhosis, in cancer and in deformity due to local constriction. In the so-called corset-liver there may be more or less of an oval projection of the right lobe extending downward several inches below the infracostal line, or in extreme cases, as far as the level of the umbilicus. A slender tongue-like extension of the liver below the right costal border (Riedel's lobe) is occasionally appreciable in cholelithiasis perhaps attended by a physical enlargement of the gall-bladder.

Inflammatory exudates are appreciated as imperfectly or perfectly circumscribing tubular resistances. They are almost always passive, and immovable with respiration. Perityphlitic exudation or tender masses in connection in the ileocecal region are usually resulting states from appendicitis. One can often feel a slightly tender, and perhaps not at all tender, mass due to the formation of thick adhesions caused by localized infiltration and exudation in this region. As a rule perityphlitic abscesses lie deep, and are surrounded by yielding walls which fluctuate. If, however, perforated perityphlitic abscess exists there may be a swashing and gurgling and splashing over this area, because of the gas and fluid accompanying this pain, leaving stomach tumors which are usually readily susceptible to examination, out of consideration.

TUMORS.

Tumors of the kidney usually appear in the loin and it is possible to push them forward by the posterior palpating hand. In addition to

going forward they usually remain covered by the hepatic or splenic flexures of the colon. Therefore percussion, palpation and inspection, particularly with the inflation of the colon with gas, generally shows the presence of the gut over the kidney structures. Renal tumors sometimes, but not necessarily move with respiration. Hydronephrosis gives rise to a renal tumor which is characterized by an elastic consistence and sometimes also by a change in size.

Tumors in the mesenteric and retroperitoneal glands are not met with frequently. These are characterized chiefly by their multiplicity and the rounded contour of the individual tumors, by their deep position underneath the intestines (recognized by percussion as well as by palpation), and by their etiology, for they are generally metastatic. Tubercular tumors of the peritoneum are palpated as nodular or irregularly defined resistances. Tubercular infiltrated and retracted omentum presents a very characteristic picture, which can be felt as a knobbed cord between the xiphoid cartilage and the navel running horizontally and superficially. Tumors of the bladder and tumors growing out of the pelvis are characterized by their position, which can be palpated directly by rectal or vaginal examination.

It is generally easy to palpate a dislocated or movable kidney, especially if the dislocation be marked enough for a "floating kidney." It is more common on the right side and unless tremendously displaced can be made out by bimanual palpation. A movable kidney can then be recognized as a bean-shaped, vertically-placed body between the two palpating hands. At times it is somewhat sensitive to pressure, giving the patient a sense of nausea. The deep location, generally beneath the resonant colon, the verification of the upper bluntly rounded end of the kidney, the lack of any sharp edge which could correspond to the liver border, should prevent confusion with a corset liver. Sometimes the hilum and the pulsating renal artery on the concave side of the kidney can be felt.

Diffuse enlargement of the spleen, acute splenic swelling, passive congestion of the spleen, the spleen of leukemia, Banti's disease, the spleen of intermittent fever are all very easily palpated and determined by their position, shape and mobility with respiration. A very large splenic tumor of leukemia and malaria, often shows one or more horizontal notches on the anterior edges. The splenic swelling in typhoid fever and other infectious diseases is often demonstrable only at the height of inspiration when the inferior edge is just below the costal margin.

As with the edge of the liver, if the spleen can be felt it is well to judge it as pathological. I have seen two cases of enlargement of the

spleen due to tuberculosis. In palpating the spleen it is well to stand on the left side of the patient and place the palpating right hand as high as possible upon the left hypochondrium with the fingers at the costal margin. Then exert firm pressure and attempt to feel the edge of the spleen during deep inspiration with the left finger tips. Most beginners are apt to employ too much force in palpating for the splenic edge—this causes a moderately enlarged spleen to sink below the finger tips.

Tenderness and pain in the region of the spleen are met with in perisplenitis, in infarction, abscess, acute congestion, cirrhosis of the liver and other forms of enlargement of the organ. Pulsation of the spleen has been described as a rare finding in Corrigan's disease.

Enlargement of the spleen is a pertinent sign in various acute specific infections, such as malarial fever, relapsing fever, sepsis, typhoid, typhus, acute miliary tuberculosis, tuberculous peritonitis, erysipelas, diphtheria, variola, scarlatina, pneumonia, epidemic cerebrospinal fever, acute yellow atrophy of the liver, and Weil's disease. In trypanosomiasis and in kala-azar the organ enlarges progressively and sometimes to an extraordinary dimension. In most of the primary anemias an enlargement is met with—myelogenous and lymphatic leukemia, Banti's disease, von Jaksch's anemia and pernicious anemia. Amyloid disease, Hanot's cirrhosis, syphilis, rickets, acromegaly, Pick's disease, and pancreatic cirrhosis serve to illustrate chronic diseases of which moderate splenic enlargement is symptomatic, and to these may be added certain lesions inducing venous congestion, such as cardiac disease, hepatic cirrhosis, and tumors causing pressure. In tumors of the spleen (such as hydatid, cancer, or lymphadenoma) and in abscess various grades of enlargement, generally of irregular contour, are encountered.

A distended bladder is easy enough to recognize when once it is felt. It may be confused with the pregnant uterus, with other enlargements of the uterus and with various tumors or with inflammatory exudates. To differentiate we must utilize other methods of examination, that is, the bimanual by the vagina, and especially examination after catheterization. Peritoneal friction may be appreciable to palpation over the different organs or over tumors of the abdomen. The friction can be felt as a rough grating with the respiratory and palpatory moving of the parts. It may also be appreciated by auscultation. Splenic enlargement in leukemia frequently leads to perisplenic, and cholelithiasis to perihepatic, friction murmurs.

Tumors of the spleen are recognized by their characteristic shape, oblique position, uniform dullness on percussion and en-

croachment upon the left thorax. A distended gall-bladder though to some extent flexible, does not remain so, but returns of itself to the edge of the liver when the restraint and pressure are removed (after it has been pushed downward), nor can the gall-bladder be pushed upward out of reach. In the case of an ovarian tumor, the mobility of the mass is restricted, usually to a level not much higher than that of the pelvis. The relation of the tumor to the uterus can be determined by vaginal palpation and its close proximity to the anterior abdominal wall can be proved by mapping out by percussion an area surrounded by a zone of intestinal tympany.

Gastric and intestinal tumors lack the characteristic mobility of a floating kidney, but they do not differ from a renal dullness in the loin. They can be traced to their source by inflation of the stomach or the gut as circumstances indicate.

A normal gall-bladder, unless markedly distended, is not palpable. But when enlarged it may produce a circumscribed globular bulging just below the right costal margin or lower. If the distention be sufficiently great to elongate the organ, the dimensions may be most extraordinary. Exceptionally, the gall-bladder enlarges in an upward direction, and is therefore impalpable. When filled with calculi, a crunching gall stone crepitus can sometimes be felt (rare).

Tumors of the small intestine are quite impalpable. Tumors of the large intestine, particularly malignant disease are not palpable excepting when they are of some size. Such tumors may then be met with in the right side in connection with the cecum and ascending colon, but very rarely met with at the flexures of the transverse or the descending colon. I have seen two instances of palpable malignant tumors of the sigmoid. As a rule malignant diseases of the rectum, even when situated high are not palpable through the anterior abdominal walls.

In some cases of colitis, particularly in the case of a chronic condition, there is a tenderness across the abdomen which corresponds to the transverse colon. The descending colon may be palpable throughout in instances of amebic colitis when there is considerable involvement of the submucous structure causing a stiffness of the wall of the gut. Less distinct stiffnesses of the colon may be felt in some instances of chronic colitis.

The pancreas being so deeply situated is usually quite inaccessible to palpation even when in a pathological condition. Perhaps in a very old, emaciated subject, one in whom the stomach lies abnormally low, the organ is palpable as a resisting mass running horizontally across the epigastrium. An area of increased tenderness

and less commonly an immobile tumor in the center of the right epigastric region may be felt in the event of pancreatic enlargement, due for example to acute hemorrhage and inflammation, to sclerosis or to a new growth. Acute inflammations of the pancreas are accompanied by such violent abdominal pains that examination is quite impractical.

AUSCULTATION OF THE ABDOMEN.

Excepting for the sounds heard over the uterus and fetal heart tones, or the cord murmurs, described in the Text-book on Obstetrics, auscultation of the abdomen is generally barren of results. One may meet with friction murmurs, tympanites with respiration caused by peritoneal exudation over the liver or the spleen, ordinarily palpable as well. In rare instances one may meet with friction sound in the region of the gall-bladder, or more particularly over the liver, as in cases of cholelithiasis. Similar friction sounds may be heard over other parts of the abdomen between rougher surfaces of the peritoneum. They are generally better appreciated by palpation than by auscultation, because they are best brought out by manipulation. In normal cases intestinal movements proceed so quietly that only very faint intestinal murmurs are to be heard, but in the pathologic increase of peristalsis the intestinal movements can sometimes be heard at a distance, the so-called borborygmi. The coincident presence of gas and fluid in the abdominal cavity may in moving a patient with a perforated peritonitis cause splashing murmurs in the abdomen, frequently metallic in character. The so-called "splashing" sounds have been mentioned under Palpation of the Abdomen.

In the diagnosis of intestinal stenosis and tumors I have often observed the presence of a hissing or whistling sound, sometimes heard at a distance, sometimes appreciated by the patient himself, taking place at the time the content of the gut is squirted through the stricture.

The normal intestines are the seat of a medley of liquid gurgles and sonorous and sibilant cooing sounds caused by the rush of gas through the unequal lumen of the gut. Through the transmitted parts cooing sounds are sometimes heard as hollow metallic echoes, also the bubbling and splashing of fluid within the stomach may be detected. One sometimes meets with abdominal auscultatory sounds of cardiovascular origin in murmurs of hepatic cirrhosis, and splenomegaly, bruits of aneurism and of compression of the abdominal aorta, the hollow echo of a heart murmur transmitted downward and

amplified by an air-filled viscus and by the telephonic properties of the parietes.

It sometimes answers the purpose to place the bell of a stethoscope over the pyloric end of the stomach and percuss the margins of the stomach from the intestines below up to the organ proper and from the ensiform above. It also answers a purpose at times to massage the sides of the cecum and ascending colon. Very often in the second week of typhoid fever a marked amount of gurgling sounds are heard in the right iliac fossa.

PERCUSSION OF THE ABDOMEN.

The examination of the human body by means of percussion plays an especially important part in diagnostic methods. It is surprising to see the lack of ability and *finèsse* of the average man in medicine to percuss properly and elucidate by percussion findings which are so well worth while. This is particularly true with regard to the abdomen where many organs are closely associated so that marginal percussion is difficult. To become familiar with percussion an interest must have been taken in doing it. This is the main reason why so few seem to learn it. The average student in the post-graduate schools makes a general percussion of the abdomen without any sort of a plan or perception of exactly what he is doing or without ever tempering his stroke according to the density of the organs he is undertaking to percuss. To become expert as a percusser one's head as well as the hands must be used. While the immediate, direct method of percussion by means of the finger tips is the most handy, it is nevertheless the most difficult to learn. When learned, however, it furnishes more accurate results than any of the mechanical means, provided one gets the sense of touch as well as the percussion note. A few rules may here be given: In finger percussion, the percussing finger should give a vertical blow, the pulp of the last phalanx being used. Plexor finger should be tightly applied. The stroke of percussion should be light, short and elastic, produced merely by the bending of the wrist joint, at the same time avoiding any cramped position of the hand or fingers. In abdominal percussion the greatest values are in the tympanitic notes as one passes from one hollow viscus to another, and then the carrying of the percussion from a viscus like the colon, to the next organ which gives a different note. Beginning with the liver it may be said that unless the organ is pathological, the liver in the abdomen is non-percussible. With the upper border of the liver, however, the dome of the dia-

phragm is percussible and corresponds to the position of the normal apex of the heart and the other side—namely, the fifth intercostal space in the mammillary line. As a general rule, however, the estimation of the highest point of the liver is of little practical value. If the diaphragm be high, the lung-liver boundary and the liver edge is always high. If the diaphragm is low, the reverse is true. Percussion of absolute or relative liver values is without practical service. One can easily show the difference on the one side as compared with the stomach on the other, and that is about as far as we can go. The best in this connection is that one's technique in absolute dullness in percussion is accurate. The lower edge is usually percussible, almost to the margin, although in my experience I would rather palpate for the lower edge even when it is not pathological, because almost to its lower edge the liver can be felt—so why percuss it in the abdomen? When a patient lies upon his left side the passive mobility of the lower liver edge is shown by a depression of the right lobe. When he lies upon the right side the reverse occurs, due to a rotation of the liver about a sagittal axis. The liver is more difficult to percuss in sitting or standing posture on account of the increased tension of the abdominal walls.

The upper border apparently lies higher than normal if a pathologic dullness from the thoracic organs (pleural effusion or lung consolidation) be superimposed upon the liver dullness. It is, therefore, necessary to resort to other methods of examination to determine whether it is a genuine high liver dullness or a normal liver dullness plus a pathological pulmonary or pleural dullness (X-ray). The upper border will be higher if the liver be pushed upward by increased abdominal tension. In this case the liver will ordinarily be rotated about a frontal axis, so that its inferior edge is elevated out of proportion to the lung-liver boundary and the vertical breadth of the liver dullness appears abbreviated. Pulmonary retraction will also cause a higher position of the lung-liver boundary. A mere diffuse enlargement of the liver without marked increase of intra-abdominal pressure, that is, without the liver being pushed upward, will not ordinarily cause an elevation of the upper liver percussing boundary. This is because the organ enlarges in the direction of the least resistance, namely, downward, and not against the diaphragm. With increased abdominal tension or if an enlargement of the liver be excessive marked increase upward will result—most frequently in unequal enlargement of the upper surface of the liver. Such tumor projections I have seen in abscess of a concavity and apparently in simple malformations. It is true that echinococcus cysts may also

A

B

Fig. 33.—*A, B,* Stiff finger percussion for noting limits of solid viscera.

produce a change in the convexity. Subphrenic abscesses produce practically identical percussion results with those obtained in tumors of the convexity—although the presence of gas may be noted.

More important are changes in the lower border. Leaving out of consideration the dislocation of the liver, the most important are in connection with stasis, a palpatory appreciation of alterations in the consistence (cirrhosis, carcinoma). Solid masses beneath the liver, packed intestines, tumors of the omentum, colon, of the stomach and the like, may increase the liver dullness downward. Palpation or a carefully repeated examination generally discloses the true condi-

Fig. 34.—Method of flank percussion on ascites. Patient on back, percussion dull.

tion. Conversely, intestines distended with air, overlying the liver and hiding part of it, may simulate a high position of the liver edge.

The liver is unnaturally enlarged in active and passive congestion, acute hepatitis, syphilis, fatty infiltration, amyloid disease, solid tumors, abscesses, obstruction of the bile duct and Weil's disease, leukemia, kala-azar, trypanosomiasis, malarial fever and relapsing fever. Decrease accompanies acute yellow atrophy as well as advanced Laennec's cirrhosis, in which the liver may be so small that the space occupied by it may be filled with coils of gut whose hollow tympany may entirely obliterate every vestige of hepatic flatness. The normal gall-bladder can not be felt unless it is stretched tight by its contents; a very tense gall-bladder without much enlargement may be palpable. When palpable the organ presents a smooth, rounded tumor at the margin of the ribs in the nipple line.

It is my experience that percussion of the gall-bladder is not a practical method of examination, but that if enlarged it is palpable. However, enlargements will be met with in hydrops, empyema, calculi or malignant disease, and also of biliary obstruction incident to simple catarrhal inflammation, intestinal parasites, and the pressure of enlarged glands, as in cancer of the head of the pancreas or of the pylorus.

For a long time it was believed by me that but little practical value could be gotten out of percussion of the various hollow viscera of the abdomen. I now have changed my mind, for so far as the

Fig. 35.—Same as Fig. 34. Patient on side so that the fluid sinks to the lowest level, floating the intestines up, causing a tympanitic note at the same spot that was dull before.

stomach and colon are concerned gross changes are possible of note. Generally, however, the X-ray method of examination is much better.

The demonstration by percussion of a tympanitic colon anterior to a kidney tumor is very valuable for diagnosis. Lastly may be mentioned the general tympanitic abdomen met with in general peritonitis, which abdomen is rounded, tympanitic in all locations even to absence of liver dullness. Such an abdomen is also commonly met with in pyretic conditions of the intestines just before the end in various affections; such as marked acute disturbances of the chest, particularly of an infected nature involving the pulmonary or pleural structures. One meets, also, with large areas of tympany in cases of congenital enlargement of the colon or Hirschsprung's disease. One,

too, not uncommonly meets with a condition described by me as idiopathic megacecum. Then, too, may be mentioned tumors rising out of the pelvis, such as large ovarian cysts, fibroids of the uterus, etc. which cause flatness or dullness on percussion. Ascites is easily percussible.

INFLATION OF THE INTESTINES.

Inflation of the intestines is best and easiest accomplished by means of an ordinary atomizer bulb attached to a colon or rectal tip

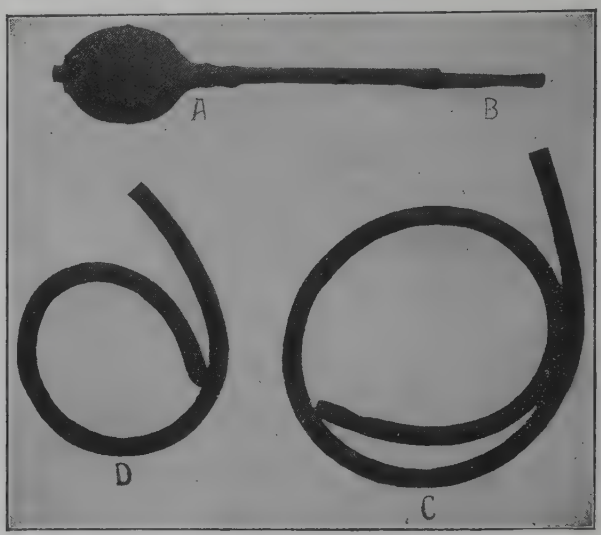


Fig. 36.—Author's assemblage for hand infiltration of the stomach or colon. *A*, Inflow bulb and tube such as is supplied with an ordinary atomizer. *B*, Short glass tube for connecting same with stomach tube (*C*) for inflating the stomach, or short colon tube (*D*) for inflating the colon.

from which jets of air are discharged into the rectum, the patient being in a prone position on his back. It is possible by this means to measure and regulate the quantity of air called for by the inflation. The tip I employ is an ordinary rectal tip from a fountain syringe set attached to the bulb. The tip is lubricated, introduced into the rectum, the patient's thighs are then brought together prone, and by squeezing the bulb it will be noticed that the sigmoid gradually distends with air. Continuing the introduction the entire colon can be filled.

It is a means of some value in detecting stenosis of the large intestine, because under ordinary conditions the jet distends the large

gut evenly. In the event of stenosis the air would distend that part of the bowel below the stricture, while above it it would remain unchanged. This is true, however, in only marked stricture, because

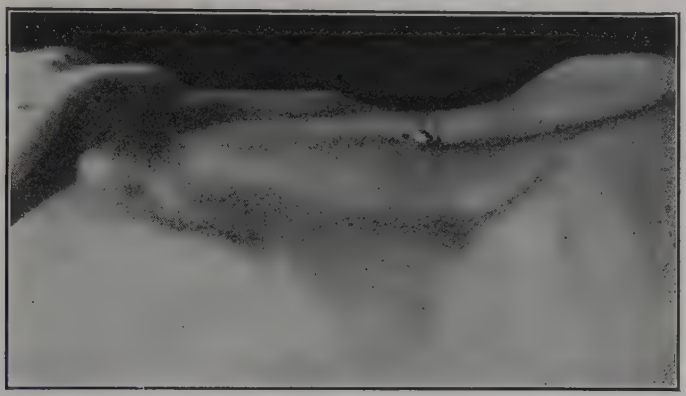


Fig. 37.—Inflation of the colon. Abdomen before air was introduced. Note the flatness of the abdomen.

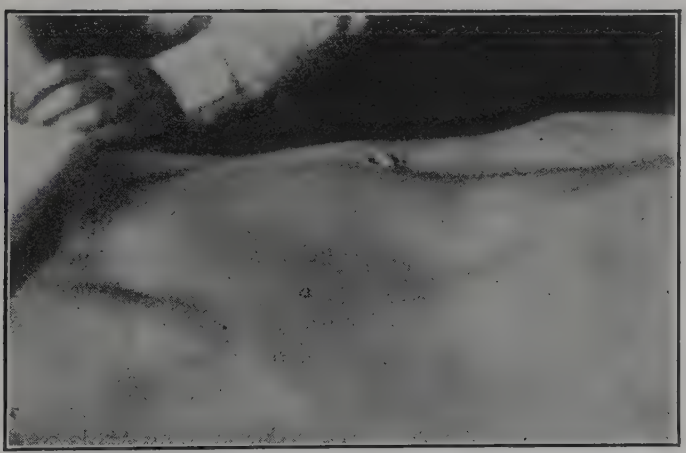


Fig. 38.—Same as Fig. 37. About 8 bulbs of air have been introluced, a few minutes being allowed before taking this picture. The entire colon is distended, rounding up the abdomen. The prominence of the sigmoid and descending colon are plainly visible. The nurse has her right hand under the cover manipulating the bulb.

in those of a mild type the air will pass through. The position of the colon can be determined by air inflation, and also the transverse width of the colon. After a considerable inflation which is accomplished by sending in jets of air until the patient complains of dis-

tention, percussion of the entire colon is very easily accomplished. Therefore it is of value in instances of dilated colon, and also in instances of enteroptosis where the transverse colon may bend down low in the abdomen. Occasionally by this means one gets a suggestion of the redundancy of the sigmoid.

For the diagnosis of the location of abdominal tumors inflation of the intestine is often of service. After inflation of the colon with air, tumors of the viscera become more distinct, while tumors of the kidney, retroperitoneal gland, etc., namely those in the posterior abdomen tend to disappear.

LOCAL EXAMINATION OF THE RECTUM.

For the local examination of the rectum I find it most convenient to use the left Sim's position for inspection of the anus and for digital examination, and the knee chest position for proctoscopic and sigmoidoscopic work. The matter of the left Sim's or right Sim's position is one of preference, according to which is the most convenient hand for the examiner to use. Most proctologists recommend that the first examination of the rectum should be made upon the patient under ordinary conditions. It is my preference that an enema be taken before the examination is made. I am rather opposed to the use of a cathartic—I recommend an enema. This insures the lower rectum being empty which is an important thing in women, because the vast majority of women have more or less fecal masses low in the rectum, and I like to finish even unto the proctoscopic examination at the time of the examination. This enema I instruct patients to take before coming to the office, and it always insures that the rectum is clear if the examination is made within one to three hours after taking it.

The patient having been placed in the left Sim's position the external parts are inspected and any departure from the normal is noted. The condition of the skin, the absence of panniculus adiposus tissue, whether the skin is normal, thickened or has a pinkish hue with lanugo hairs, suggesting tuberculosis. The external opening of a fistula, would be most easily discovered, particularly by drawing the folds about the anus away from each other. Fissures, external hemorrhoids, pediculi, and perhaps pin worms will be easily seen. You note whether the skin is thick and moist, or dry (such as would be seen in cases of long-standing pruritis). Also, it will be observed whether the sphincter is normal, relaxed or spasmodic, whether there is any evidence of inflammation, abscess formation, or whether external thrombotic hemorrhoids exist.

The patient should bear down and the condition of the anus be observed. This procedure is often all that is necessary to make a diagnosis; especially in the case of fissure, hemorrhoids, fistula and hypertrophied papillæ. A polypus with a long pedicle, situated near the opening might come into view, as will also prolapse of the rectum, partial or complete.

The next point is the palpation of the tissues about the anus in which instance it is sometimes possible to trace a fistula without the aid of a probe; to outline an abscess and to note painful points. By palpation directly upon the anus it is possible to get a fair idea of the condition of the sphincter. Where there is hypersensitiveness of the patient, a digital examination may be impossible without an anesthetic.

Digital Examination: The examiner employs the index finger, well lubricated and of course with a carefully cut nail. The finger may be protected by employing the ordinary thin rubber finger top or a thin rubber glove, thus protecting the back of the hand as well. Separating the folds of the buttocks with one hand the finger should be introduced slowly and carefully with a gentle screw-like motion so as not to cause pain or injury. Beginners commonly make the mistake of not following the axis of the rectum after introducing the finger, especially when they are trying to palpate very high. The anatomic position of the rectum should be remembered—namely, the direction is first a little forward from the anal opening, then backward into the hollow of the sacrum, and finally to the left toward the sigmoid flexure. While advancing the finger one must remember to keep it always in the middle of the furrow formed by the mucous membrane. If feces be present it will be the best guide as to direction in which to advance the finger. The height to which the palpating finger may reach depends upon various factors. An examiner with short thick fingers and the patient excessively fat are the chief obstacles. Sometimes the pain caused by the introduction, as in a case of spasmodic contraction of the internal sphincter will prevent an effective examination. Digital examination reveals first of all the more crude anatomic changes, carcinoma of the rectum low down, other tumors and ulcerations of the rectum, rectal polypi which are not situated too high up; large internal hemorrhoidal masses will also be felt (but unless thrombosed it requires considerable experience to differentiate them with certainty from normal inequalities of folds of the rectal mucous membrane). It is always well to keep away from any painful areas. Particularly is this true when sweeping the finger around the rectum. By doing this the condition of the

prostate may be noticed and in the female the cervix uteri is easily felt, particularly when the uterus is retroverted or enlarged. The latter is an important point because not a few instances of trouble about the rectum are met with (mainly in the way of subjective symptoms) in cases of retroverted or retro-uteri. At the same time while the finger is in the rectum a prolapse or intussusception of the sigmoid may most easily be made out. Any redundancy of the rectal mucous membrane, hypertrophy of the valves of Houston, presence or absence of fecal matter, malignant tumor, stricture, benign growths or any abnormality within the reach of the finger can be determined. Of course, about all that can be made out are lesions in the first three and a half inches of the lower end of the rectum. On the withdrawal of the finger, the index finger of the other hand may be placed upon the coccyx and this grasped between the two fingers, moved around,

Fig. 39.—Brinkerhoff rectal speculum.

and any abnormality of its condition or painful point noticed. Hypertrophied papillæ, abrasions of the mucous membrane, abscesses, the internal opening of a fistula, can all be easily made out by the educated finger. Finally, as the finger is withdrawn, the condition of the sphincter muscle may be determined again, and if there are any painful points it will contract spasmodically, the latter suggesting local inflammation. Finally, with the withdrawal of the finger it should be noticed whether it is slimy, or whether purulent or bloody masses adhere to it, this helping in the diagnosis, and perhaps requiring a microscopical examination of the adherent matter. A microscopic examination of the adhering material will sometimes aid in the diagnosis of tubercular or dysenteric changes, and perhaps be of some service in the examination of the feces, particularly if the rectum has just been cleaned of fecal matter by an enema. The introduction of the hand for the purpose of diagnosis as suggested by Simon of Heidelberg, is only mentioned to be discouraged.

With the aid of the so-called rectal speculum the examiner can see the inner surface of the rectum directly. There are various forms

of rectal specula, running all the way from those like vaginal speculum, tubular, bivalve of duck-bill shape, to those of many valves and sides. The one I most commonly use is the plain Sim's vaginal speculum of small size, having the traction posterior. This gives a very good view of the portion of the rectum in front of the concavity of the bill-shaped end, and it is possible to get a view of the posterior surface of the rectum. The next instrument and one which is becoming increasingly popular—is the Brinkerhoff rectal speculum; it is very practical and satisfactory for certain kinds of rectal work. It recently has been improved by the incorporation of an electric light for direct illumination. This instrument in the instance of a

Fig. 40.—Hanes rectal table. While the knee-chest or left-side positions may be used for the endoscopic examination of the rectum and sigmoid, the inverted position of Hanes is far more satisfactory and when many cases are being examined or treated locally, saves time as well. This position may be accomplished without a special table by the patient resting with a shoulder on each of two chair seats, the head hanging down between them. The above suggested table serves excellently for the purpose.

normal or relaxed rectum or sphincter is possible of being twisted about with comparative ease. Where the sphincter is quite tight it is sometimes necessary to reintroduce the slide, returning the instrument in this way so as not to injure the mucous membrane of the anal opening. The third instrument is the rectal speculum suggested by Cook which possesses the advantage of a slightly better view, particularly in cases where the sphincter is of considerable size. The blades of this instrument are narrow and a rather even illumination is obtained with a powerful white light showing the tissues in their natural colors.

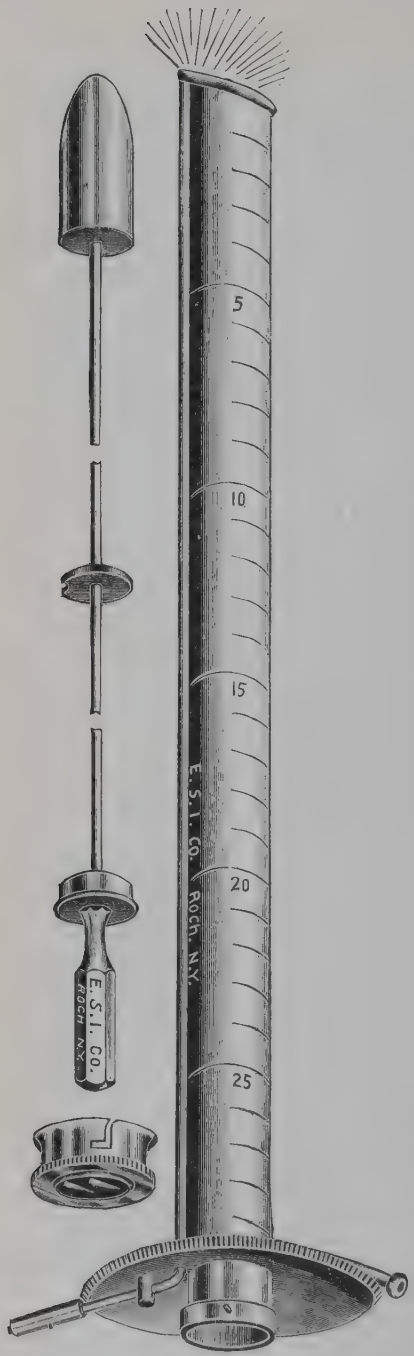


Fig. 41.—Author's proctoscope, and the longer tube, proctosigmoidoscope.

Altogether, the examination with the speculum is difficult on account of the limitation of the field of vision and the fact that the parts are usually in an abnormal position and under abnormal tension so that the interpretation of the findings may be difficult. However, such conditions as palpation failed to reveal, as inflammatory congestion, superficial ulcers which are not palpable, are immediately brought into vision by the use of one of these instruments.

The results of the ocular inspection of the rectum have markedly improved since the advent of the modern proctoscope and it is quite surprising to me that, considering the number of proctoscopes sold, more general use of proctoscopy is not made, and further surprising that the average person who possesses this instrument does not know how to pass it with ease. This is important because to the average individual the passage of the proctoscope is most distressing unless it is done properly. And when it is properly done the attendant distress is but very little. Thorough lubrication is very important, and it is my habit to lubricate the interior of the anus before the tip of the instrument is introduced (petroleum jelly is best). The patient should be in the knee-chest position on a table, with the buttocks about on a level with the examiner's face. Before the introduction of the obturator of the instrument, it is well to

pass the lubricated left index finger slightly within the rectum. The patient is then asked to bear down slightly and the obturator of the proctoscope is passed, the tip of the obturator resting on the inner surface of the index finger. As the instrument is passed in, the auxiliary tube comes along after the obturator, and the index finger is gradually withdrawn. The end of the instrument being on the inside of the internal sphincter, connections are made between the light carrier and the rheostat, and the light, which has been tested in advance, is turned on. The obturator is now withdrawn and the instrument is passed from this on entirely by sight so as not to injure any part of the mucous membrane on the way upward. The plug and glass window which closes most instruments is attached, and jets of air are passed into the rectum and sigmoid if the mucous membrane encroaches too closely upon the advancing tip. When the rectosigmoidal junction is reached, the sigmoid which adjoins the rectum turns to the right or the left; in my experience, more frequently to the right. It is at this point that difficulty is encountered, even by the most experienced, and it is sometimes impossible to go beyond this point without great danger of material damage. However, a little manipulation and skill sometimes permits the instrument to be passed without difficulty. The reason for this is that where the sigmoid joins the rectum it forms an acute angle and over this one of Houston's valves will fall obscuring the direction of the gut from the examiner. How far the instrument can be passed when it enters the sigmoid depends upon the length of the bowel. At no time, except where the mesentery of the sigmoid is very short and where very little sigmoid exists, can the instrument be passed above the apex of the sigmoid. If the instrument continues to pass after this, it is because the sigmoid and mesosigmoid are very long; in a few persons, it almost reaches the diaphragm. I have been able in instances to make diagnosis of adhesions at the brim of the pelvis, which will be met with in connection with colonic adhesions, particularly in connection with constipation, where advancing to that point it is impossible to advance further. However, it is possible at times to pass the instrument until its entire length has been introduced, and even to feel the end of the instrument in the abdomen.

Since the introduction of the proctoscope, instead of the limited view obtained by the ordinary rectal speculum where orientation is very difficult on account of prolapsed folds of mucous membrane, a clear, free view into a stretched open cavity is obtained on the walls of which, with the advance and withdrawal of the instrument, not the slightest changes can escape observation. One can with com-

parative ease examine directly 20 and in many instances, 30, 35 and 40 centimeters of the lower bowel quite beyond the possibility of digital examination.

Since the introduction of the original instrument numerous forms and modifications have been advanced by various workers in proctology. Not specifying the various forms, simply confining myself to those that are the most popular, I mention the proctosigmoidoscopes of Tuttle, Lynch, Beech and Strauss. My experience until recent years has been mainly with the use of the Tuttle instrument. This possessed some disadvantages. In the first place the applying end of the tube is sharp and the mucous membrane was sometimes injured by it. The handle which was adjustable was inconvenient, and the screw end was more or less of a nuisance, it constantly unscrewing. Then what was most distressing of all was that the window fitted snugly into the proximal end of the tube, making its withdrawal very difficult, and this is sometimes most distressing, particularly when one desires to remove the window during the course of the examination. If a light on the proximal end is desired the use of the Lynch instrument is to be recommended. In my experience, however, the distal end lighting is much to be preferred, giving a stronger light with the background of a dark tube; and a better view of the mucous membrane is obtained.

To obviate the danger and inconveniences incident to the use of the instrument, I have devised one which possesses the following features: The tube is marked off in centimeters so that the depth of insertion may be seen. To prevent injury to the mucous membrane. the distal edge is rounded this being the first instrument in which this was done. The air-tight window is not pushed in but fits over the proximal end of the tube and may be slipped on and off by a slight turn. The disc shield is basin shape, large and milled. The auxiliary tube for dilatation opens directly into the main tube. The light carrier is held in place by a set screw on the disc so that it cannot be pulled back by inadvertent traction on the conducting cords. The value of this instrument has been greatly increased by the more powerful illumination of the tungsten lamp with which it is now equipped. The instrument is made up in the proctoscope size, 12 centimeters long, and proctosigmoidoscope size, 30 centimeters long. I would recommend the use of a single dilating bulb which is what I use instead of the double one as supplied in the Tuttle and Lynch instruments.

By the use of this instrument the diagnosis of carcinoma of the rectum is made at a glance, and other very important conditions are

easily recognized. Other possible diagnoses may be enumerated as deep hemorrhoids, which are not accessible to palpation and which may cause obscure intestinal hemorrhage and anemia; the existence of an intestinal rash and easily abrased mucous membrane, responsible for obscure bleeding; benign growths which to the digital examination give the impression of malignancy; tracts of ulcerations, particularly those in connection with the bacterial ulceration, hemorrhagic, tubercular, syphilitic and follicular; the internal point of entrance of fistulæ, cryptitis and proctitis; stricture of the rectum and lower colon; rupture, injury and wounds of the rectum; inflammations of the colon and rectum, particularly as seen in cases of hypertrophic and atrophic proctitis; and the opening of a diverticulum may be observed, high in the rectum and particularly in the colon. One occasionally encounters the discharge of a diverticular abscess taking place within the rectum; pigmentation of the bowel which is significant in connection with intestinal toxemia; and various forms of benign tumors and polypoid formations.

Thus it will be seen that the proctoscopic examination of the rectum and lower colon is very valuable in connection with the diagnosis of entero-colonic conditions, particularly the chronic forms of inflammation and ulceration. I do not see how gastroenterology can be practised without its use, and I am sure that one who confines his work to the rectum and anal regions can not but receive great benefit of a practical kind in the suggestions that must come in connection with the treatment after an observation of the condition of the mucous membrane.

TESTING THE INTESTINAL FUNCTIONS.

Inspection and auscultation of the abdomen will give a suggestion at times of the motility of the intestines, but are not dependable methods. The manner of defecation and the examination of feces will also aid. While in a general way the passage of flatus is more indicative of the production of gas in the intestines, at the same time a free and abundant passage of gas indicates good, intestinal motility, while a complete inability to evacuate any flatus suggests some impairment in intestinal motility. This latter condition is important in types of ileus where solid or fluid evacuations still take place, but where the absence of evacuated gas presents such a contrast as to furnish a very significant symptom of disorder.

There is a time-honored method of testing the rate of transit through the intestinal canal by taking such substances as charcoal, lycopodium or carmine, and watching the stools to determine the

interval required for these substances to appear. Charcoal will be recognized by the black color, lycopodium by the characteristic tetrad shape of its spores under the microscope, and carmine by the reddish color it imparts to stools. Of course, in all of these tests the rate of transit through the gastro-intestinal canal, the gastric motility must be taken into account in figuring the results.

A careful examination of the feces will aid in determining the intestinal chemistry, especially with reference to the utilization of food. But the disadvantage of this is that ordinarily we do not consider the influence of the length of time the food constituents remain in the intestine. However, there is no doubt that it represents the most valuable means at hand for the testing of intestinal functions, and will be mentioned more fully later in the chapter.

More or less ingenious procedures have been devised in the past for testing intestinal digestion. First, Unna's keratin coated pills which may only be mentioned as not having proved successful; next the so-called glutoid capsules which are filled with various substances, usually those containing iodine are best adapted for the purpose. Iodoform has given fairly accurate results in this connection. It is absorbed both in the stomach and the intestines in a very short time—within from one-quarter to one and one-quarter hours. After the ingestion of 0.15 gram of iodoform a marked iodine reaction can be obtained in the saliva or in the urine, especially so in the former, with chloroform, sodium nitrite and sulphuric acid. One of the substances that may be contained in the glutoid capsule is salol, which appears in from one to one and a half hours after the ingestion of 0.5 gram, when salicylic acid can be demonstrated in the urine with ferric chloride as salicyluric acid. One of the objections to filled glutoid capsules is their varying degree of hardness. For diagnostic purposes the capsule of maximum hardness is requisite.

In order to make the conditions as uniform as possible in regard to the length of time that the capsules remain in the stomach, and in regard to the degree of digestive stimulation, it is advisable to administer them with an Ewald test-breakfast. Under normal conditions the ideal reaction may be expected to appear in the saliva and urine and the salicyluric reaction in the urine in from four to six hours.

Three hours after the administration, and at regular intervals thereafter, the patient expectorates saliva, or better, voids urine in numbered beakers. These specimens are examined later for the presence of iodine or salicyluric acid according to the tests indicated. I have found that the elimination by the saliva is so irregular that it is of no practical value, and that the urine tests are not very much

more to be recommended. However, where the voiding of the substances strongly suggest a normal condition of affairs, the use of the testing methods are worth the while. But in my experience there is so much irregularity met with in normal people that I have quite given up the use of the glutoid capsule. One cannot tell whether the capsules are dissolved, either by the gastric juice or by some chemical agency in the intestines other than the pancreatic secretion. Therefore after the tests are completed, even though they are normal or perhaps abnormal, one is left quite in the dark as to their practical significance.

The next test to be mentioned is that suggested by Schmidt of the use of cell nuclei which are supposed to be digested by the pancreatic juice and not in the stomach. Here a small square of beef is cut into one-half centimeter cubes, and hardened in absolute alcohol. These are put into tiny bags of silk gauze, so that they may easily be recovered from the feces, and set aside in alcohol. A few hours before use the bags are immersed in water. The patient swallows these bags upon several successive days during which he eats a definite diet. The bags being recovered from the feces, they are washed and their contents, which are chiefly muscle residue, examined for nuclei after making fresh preparations with acetic acid and staining dry smears with methylene-blue. According to Schmidt, if the pancreatic function be not completely lost, the nuclei may be digested. So many errors are possible in this method of testing the pancreatic function that I have quite given up the use of these bags. There are instances of the bags remaining too long in the stomach or being ejected too rapidly. The hastening of the bags through the intestinal canal in cases where the bowels are quite normal, or even more so in diarrhea interferes. And then many times I have met with experiences where the examination of the muscle fibers in the bags strongly suggested a normal pancreatic function only to find at operation that the pancreas was distinctly diseased and therefore deficient.

Following the demonstration that under conditions of straining it is possible to force the duodenal contents into the stomach, and by aspiration of the stomach and examining the content for proteolytic, lyptolitic and dyastatic action to prove the presence of pancreatic juice, which is always strongly suggested because of the staining of the stomach contents with bile, there took place the test of Volhard in which it was possible to cause a regurgitation of pancreatic juice and bile into the stomach if an abundance of fat be ingested. He introduced 200 cubic centimeters of olive oil through

a tube. One-half hour later he withdrew the fluid, which could be readily separated from the oil either by the use of a pipet or a separating funnel. The advantage of this over the Boas method is perhaps that the pancreatic juice obtained in this way is more constant and probably of a physiologic character corresponding to the stimulation employed. Volhard was then able to demonstrate tryptic action in 86 per cent. of his cases by the casein method of examination.

One of the important facts to keep in mind with these methods of examination is that obviously there is no alteration of the intestinal juice by that of the gastric, and since they are both obtained in abnormal ways, I have quite given up these methods of examination.

Next should be mentioned the fibrin method, advanced by Arthus and Huber, in which a solution of fibrin and sodium fluorid is made, and after filterization is used to demonstrate the presence of trypsin. This to me is a most fallacious method and is mentioned to be discouraged.

EXAMINATION BY DUODENAL TUBE.

Interest of late years has centered in attempts to diagnose abnormal conditions of the pancreatic gland, biliary secretion and the secretions of the duodenum by means of the so-called duodenal tube. Einhorn, Gross, Jutte and others have written extensively upon the subject of a direct method for observing the secretions of the pancreas. My experience is limited to the Jutte tube which I believe to be the best for the reason that it is easily introduced into the stomach and duodenal secretions are commonly obtained in not more than ten minutes from the beginning of the introduction. This tube follows the usual plan of tubes as to the caliber of the tubing, but a small wire mandrin is used and the end of the tube is small so that it quickly passes through the pylorus. After the passing of the tube into the stomach the mandrin is withdrawn, the patient lies on the right side, drinking small quantities of water, and an aspirating bottle is used to observe a return which when viscid or strongly alkaline represents that the end of the tube is in the duodenum.

The duodenal juice is usually golden yellow, viscid, slightly acid due to gastric content, or neutral. Amounts of from 10 to 40 cubic centimeters can often be recovered. The stomach content is milky white and strongly acid. In testing for the enzymes, the specimens are generally diluted with distilled water, using twice the amount. A part is immediately made slightly alkaline with sodium hydrate solution, this serving for the alkaline protease test; the remainder is used for testing amylase and lipase. The chemical methods for analyzing the duodenal ferments are as follows:

For Amylase: One cubic centimeter of the duodenal juice is tested against increasing amounts (½ to 6 cubic centimeters) of 1 per cent. soluble starch solution, the volume in each test tube being made up to 10 cubic centimeters with water. After an incubation of one hour, the persistence of the starch is tested for by adding a small excess of Lugol's solution. In a series of test tubes the disappearance of the starch reaction is read, and the number of cubic centimeters of starch solution used, multiplied by the dilution, can be accepted as the factor. This method, as suggested by Crohn,[1] is better than the Wohlgemuth method.

For Lipase: To 10 cubic centimeters of distilled water are added 1 cubic centimeter of the material to be tested, 1 cubic centimeter of ethyl butyrate, 1 cubic centimeter of toluol, and a drop of phenolphthalien solution; the whole made up to 25 cubic centimeters and neutralized. After shaking for fifteen seconds or more it is again brought to the neutral point. A control test is prepared with boiled duodenal contents. After twenty-four hours incubation both flasks are titrated, and the amount of acid in the control subtracted from that in the test flask, and the result multiplied by the dilution.

For Protease (alkali): Mett tubes, cubes of coagulated egg albumin, Fermi gelatin tubes and the Gross-Fuld casein method can be utilized.

It is best to remove duodenal content in the morning and immediately ice the returns, they to be examined in the afternoon. It is found that an acid reaction is preferable for preserving the fluid for both amylase and lipase tests. In acid reaction these ferments may be preserved for from twenty-four to forty-eight hours in undiminished strength. In alkaline reaction an apparent auto-digestion takes place, probably due to the presence of trypsin. Lipase is best maintained in the faintly acid medium, but trypsin is most active in an alkaline.

The duodenum normally contains at least two proteases, trypsin and erepsin, the latter secreted by the duodenal mucosa, as well as by the pancreas. Erepsin as a rule does not attack the coagulated albumin tests. Casein, however, is digested by erepsin, although its action upon casein is only faintly proteolytic.

The above mentioned tests may be used for the examination of stools. There is some confusion as to whether a slight digestion is due to erepsin and where when the proteolysis is complete, the results may be interpreted as due to trypsin. These methods of analyzing the duodenal content for these enzymes, as well as the stools have not advanced sufficiently to make deductions positive. It

will be noticed in engaging in this work that quantitative estimates of the strength of pancreatic ferments obtained from the duodenum of a normal man vary within such wide limits that interpretation of results are practically not worth much. The presence of the three ferments can easily be demonstrated in an active state; lipase, however, may be absent occasionally. In the observations of Crohn a case of acute pancreatitis displayed a diminution in the ferments with the exception of lipase. In cases of cholelithiasis ferments were found in an active state in the duodenum, with a wide range of variation. Cases of obstructive jaundice are influenced according to the patency of the pancreatic duct, which when open, invariably shows the presence of active enzyme. Cases of hypertrophic cirrhosis of the liver showed active ferment analyses. A case of achylia gastrica which showed the absence of both pepsin and rennin as well as all trace of acid, showed active pancreatic secretion. Cases of diabetes mellitus did not show any variation from normal figures. It must be remembered by the reader that these ferments from the duodenum show the greatest variability in strength and may occasionally be absent even in health. The protease is the most constant and is always present. There is value in this method of examination in some cases of acute pancreatitis or in obstructive conditions of the pancreatic duct or the biliary duct by simply noting the absence of bile in the return fluid. But beyond this point there is practically nothing to encourage further research or practical utilization of this method of examination. In the rare cases of carcinoma arising in the ampulla of Vater this method, as shown by Crohn, possesses diagnostic value. The same may be said of neoplasms arising in the duct of Wirsung or from the duodenal surface obstructing these ducts. Such growths may come from ulcer and the obstruction may be diagnosed early by the use of the duodenal tube.

EXAMINATION BY TEST DIETS.

The most valuable method at present for testing the intestinal functions is the normal one of a test diet. The first efforts in this direction were made by the diets of Strassberger and Schmidt in their diets Nos. 1 and 2. Very shortly after their advancing these diets it was manifest that they were far too complicated for ordinary use and unnecessarily so. Efforts were then made in various directions to simplify the diets, and these have been more or less changed by various observers in this field. One of the first modifications was made by Schmidt himself, in what he designated as a general test-diet which is as follows:

General Test Diet.

In the Morning.—½ liter milk, tea or cocoa (if possible with much milk), together with 1 roll and butter and 1 soft-boiled egg.

Breakfast.—1 dish of oatmeal-gruel cooked in milk and strained (salt or sugar permissible); gruel or porridge may, under certain conditions, also be given.

At Noon.—¼ pound finely chopped lean beef, broiled rare, with butter (the interior raw), along with it not too small a portion of potato broth (well strained).

In the Afternoon.—Same as in the morning, but no egg.

In the Evening.—½ liter milk or 1 plate of soup (as in the morning), together with a roll and butter and 1 to 2 soft-boiled eggs (or scrambled).

It is obvious that the above is too general to permit of accurate work and Schmidt realized this himself when he advanced the so-called Detailed Test-Diet for Clinical Purposes and Quantitative Analyses:

Detailed Test Diet.

In the Morning.—0.5 liter milk (or if milk does not agree, 0.5 liter cocoa prepared from 20 grams cocoa-powder, 10 grams sugar, 400 grams water, and 100 grams milk), with 50 grams zwieback.

In the Forenoon.—0.5 liter oatmeal-gruel prepared from 40 grams oatmeal, 10 grams butter, 200 grams milk, 300 grams water, 1 egg (strained) and some salt.

At Noon.—125 grams chopped beef (raw weight), broiled rare with 20 grams of butter, so that the interior still remains raw. To this 250 grams potato broth made of 190 grams mashed potatoes, 100 grams milk, and 10 grams butter and some salt.

In the Afternoon.—As in the morning.

In the Evening.—As in the forenoon.

The diet is given for three days, and sometimes longer—at any rate, until a stool is obtained, which comes with certainty from this diet. The patient does not need to take any special means in order to mark off the normal excrement from the excrement of the former diet, for the normal excrement can generally be recognized at once by its uniform consistency and its lighter color.

Several have criticized the Schmidt diet because of the milk it contains on the reasoning that milk is not the proper substance to use in testing out intestinal function. My belief concurs exactly with Schmidt in that even if milk does create a disturbance that it is well for this disturbance to take place so that one can glean from the findings of the examination of the stool specimen just what the change from normal would be.

The diet that I use in my work, and which I have employed in some thirty-seven hundred instances and have found practical and complete and easily followed by anyone, those up and around as well as those in the hospital, is the following:

The diet mentioned below is to be adhered to strictly for three days. The stools of the first forty-eight hours after its institution are of no value for examination. If constipation exists the bowels are to be moved by injection. After this time, providing the bowels have moved well, the entire next stool is to be sent, no purges being taken in the meantime, but a glycerin suppository or an enema can be employed to obtain the stool required. A covered small tin can or a glass jar can be used as a container. A twenty-four hour collection of urine should accompany the stool, this being started at the end of the forty-eight hours after the institution of the diet. Take no medication by mouth during the three days.

Morning.—Two thin slices of well baked bread with butter liberally applied; 1 pint of oatmeal-gruel, made of about 40 grams or 1⅓ ounces of oatmeal; 10 grams or ⅓ ounce of butter; 200 grams or 6⅔ ounces of milk; 300 grams or 10 ounces of water (all strained), 1 egg cooked in any form.

11 A.M.—Milk, 1 pint.

Noon.—A good sized piece of steak or roast beef, chopped or cut into very fine pieces (about 120 grams or 4 ounces) and served on a slice of toast; 1 bowl (about 250 grams or 8⅓ ounces) of mashed potato with 20 grams or ⅔ ounce of butter.

4 P.M.—Milk, 1 pint.

Night.—Same diet as breakfast.

Water may be taken as desired.

It will be observed in the above that I demand a twenty-four hour specimen of urine which is carefully examined for the quantity of phosphates and sulphates, both the preformed and organic forms, and the presence of indican, urorosein, the quantity of oxalic and uric acids, etc., for I believe that these additions to this method of testing intestinal functions are most valuable.

Test Diet for a Child.—The diet mentioned below is to be adhered to strictly for three days. The stools of the first forty-eight hours after its institution are of no value for examination. If constipation exists the bowels are to be moved by injection. After this time, providing the bowels have moved well, the entire next stool is to be sent, no purges being taken in the meantime, but a glycerine suppository may be employed to obtain the stool required. A covered small tin can or a glass jar can be used as a container. A twenty-four hour collection of urine should accompany the stool, this being started at the end of the forty-eight hours after the institution of the diet.

Morning.—One thin slice of well baked bread with butter liberally applied; ½ pint of oatmeal-gruel, made of about ⅔ ounce of oatmeal; 5 grams or ⅙ ounce of butter; 100 grams or 3⅓ ounces of milk; 150 grams or 5 ounces of water (all strained). One egg cooked in any form.

11 A.M.—Milk, ½ pint.

Noon.—A good sized piece of steak or roast beef, chopped or cut into very fine pieces (about 60 grams or 2 ounces) and served on a slice of toast; 1 bowl (about 125 grams or 4⅛ ounces of mashed potato with 10 grams or ⅓ ounce of butter.

4 P.M.—Milk, ½ pint.

Night.—Same diet as breakfast.

Water may be taken as desired.

The Roentgenological method for noting rates of transit through the intestinal canal has been dwelt upon in connection with intestinal movements (normal), and the Roentgenological examination (abnormal) in the following chapter.

THE FECES.

The feces are often regarded as representing the undigested or indigestible constituents of the food which have escaped solution and absorption in their passage through the alimentary canal. This is not correct as applied to man or to the carnivora. While a certain amount of food taken by mouth is found in the feces, the feces more completely represents not derivations from the food products, but from the alimentary canal itself. This is shown by the fact that on analyzing the feces no soluble carbohydrates or proteins, albumoses or peptones or amino-acids are to be found. Microscopic examination of the feces after a meal of meat reveals no trace of striated muscle fibers, unless the passage through the intestinal canal has been very much hastened, as in the case of a diarrhea. As long as vegetables or coarsely ground cereals are excluded from the diet, the nature of the feces does not alter the chemical constitution or appearance of the feces. With almost any diet, except that containing large amounts of cellulose, the composition of feces is about as follows:

Water	65 to 67 per cent.
Nitrogen	5 to 9 per cent.
Ether extract	12 to 18 per cent.
Ash	11 to 22 per cent.

The ash consists chiefly of lime and phosphoric acid with some iron and magnesia. The ethereal extract contains fatty acids and a small amount of lecithin. Neutral fat is found in very small proportions. The feces also contains small quantities of cholalic acid and its products of decomposition, dyslysin, and coprosterin (a body allied to cholesterin), and a certain amount of purine bases consisting of guanine, adenine, xanthine, and hypoxanthine. The material basis of the feces seems to be to a great extent desquamated epithelial

cells from the intestinal wall, and bacteria, of which countless numbers, chiefly dead, are present. It has been reckoned that as much as 50 per cent. of the feces may consist of dead bodies of bacteria, but this is probably a little high as regards normal man.

Very different, however, is the composition of feces if the food contains a large amount of cellulose. Not only does the ingested cellulose pass unchanged into the feces, but large quantities of other substances enclosed in the cellulose walls may also escape digestion and absorption. Moreover, the increased bulk of the undigested residue stimulates peristalsis, and thus quickens the passage of the food through the gut to such an extent that the digestive ferments have no time to exert their full action on the digestible constituents of food. The influence of the character of the food is well illustrated by a comparison of the amount and composition of the feces on different kinds of bread. (Rubner):

Kind of Bread.	Weight of moist feces.	Weight of feces dried.	Percentage of ingested food.	Nitrogen (grams).
Bread from fine flour	132-7	24-8	4-03	2-17
Bread from coarse flour	252-8	40-8	6-66	3-24
Brown bread	317-8	75-79	12-23	3-80

The indigestible cellulose in the food is not without value. It has been shown that the peristaltic contractions of the intestine are roused primarily by the mechanical stimulus of distention. If the food is capable of entire digestion and absorption the amount of feces formed is limited to that produced by the intestinal wall itself. The small bulk exercises very little stimulating effect on the intestine, and the movements, especially in the absence of the mechanical stimulus are determined by muscular exercise. The presence of a certain amount of cellulose in the diet may therefore be of considerable advantage by giving bulk to the feces and ensuring proper regular evacuation of the lower gut.

EXAMINATION OF THE FECES.

There is considerable variation in the frequency of bowel movements even in health. Some healthy people have movements of the bowels only once in two or three days, and others have several each day. Just what the pathologic and physiological limits are is hard to say because they are different in different individuals. Of course, to have a standard to go on it is best to have it figured on the basis of one or two bowel movements in every twenty-four hours. The term "constipation" is usually restricted to a condition of infrequent movements which is associated with certain other difficulties and

which does not bear a proper relation to the quantity of food taken. In the same way the term diarrhea is employed when the movements are not only frequent but also liquid.

The normal consistence and shape of feces is well known—hard in constipation and fluid in diarrhea. Between these extremes are all sorts of intermediate forms. The small fecal balls which occur in intense constipation, from the tightly packed fecal matter becoming friable, are especially noteworthy. On the other hand, scybala may be of very unusual volume in constipation if a large quantity of feces stagnates in the rectum and distends it mechanically. In intestinal stenosis but a short way above the anus the fecal masses have a diminished transverse diameter; but if the stenosis be high up, their shape changes again below the obstruction.

The normal color of the stools of an adult is dark brown. This color is not due to biliary pigment, but to its secondary products—urobilin, etc. Infants' stools are normally light yellow or golden yellow, because they contain unaltered bilirubin.

The color of the feces varies more or less according to the nature of the food and the status of the bacteriology in the gut. A milk diet and much fat gives a light color to the stools; abundant consumption of red wine, blueberries, blackberries, or black cherries, gives them a dark color. Food rich in chlorophyll (vegetables) may produce a green or an olive shade stool. Chocolate, and especially cocoa, causes a noticeable red-brown color. Drugs may also change the color of the feces. Calomel is apt to color the stools greenish, due to the transformation of the bile-pigments into urobilin, and also to the sublimate derived from the calomel which changes the bilirubin into biliverdin. After the use of bismuth the stools are generally of a blackish color, which is due to the reduction of the bismuth salt into bismuth suboxid. The characteristic color of the stools after taking iron is well known.

Under pathological conditions the stools may be abnormally colored by the addition of blood. The stool is abnormally light in color in acholia, from the deficient production of bile with the absence of icterus, and in retention of bile due to occlusion of the biliary passages in icterus. Such stools present a peculiar grayish-white appearance—the so-called clay-colored stool, which color is due not only to the deficiency in the amount of bile, but also to an abundance of unabsorbed fat.

In diarrhea the movements are generally light colored, because the pigment is distributed through a much larger volume, and because of the unchanged bile pigment in the stools. Sometimes undigested

food-particles are found in the stools, which are of no diagnostic significance, particularly in connection with such things as seeds, stones, skins of fruits, and large masses of cellulose substances. This is of not much importance excepting in connection with certain "lientery" conditions, where if there was a conspicuous amount of food passed in an unchanged state it would have significance in the way of denoting a lack of digestion and absorption.

The odor of normal human feces is well known. It is due chiefly to indol and skatol, and perhaps also to methyl mercaptan. The fecal odor is much more pronounced upon a meat than upon a vegetable diet, due to the fact that there are larger quantities of protein for bacteria to act upon and the production of various sulphate gases.

There may be a noticeable admixture of mucus in the stools, which will be noticed here as in other excreta usually by its peculiar consistence and transparency. Sometimes this mucus is passed in shreds or rolls and sometimes passed as true mucin, that is, in a fluid state. Usually it is in long pieces, rolled that way by the intestinal peristalses.

Even a normal stool contains some mucus, which, however, is only demonstrable chemically. We must not confuse this with bits of swollen vegetable tissue, especially that from fruit, which are frequently found in a normal stool and can be easily recognized microscopically by their cellular structure. Large shreds of mucus in thin stools are found, especially in catarrh of the large intestine, and fluid mucus in irritative conditions of the lower part of the large intestine or the rectum.

The stool may contain blood, which will exhibit itself in different ways. It may be recognized macroscopically by a red or blackish color, but it may not be visible to the eye and has to be searched for by one of the various chemical tests, of which the most valuable in my experience is the aloin test. Solid feces coated externally with blood would indicate a hemorrhage in the lower portion of the intestine, where the feces is already solid and formed, such as may be seen in hemorrhoids. Solid feces evenly tinged throughout would point toward a hemorrhage in the stomach or the upper part of the intestine. Even in liquid stools the admixture of blood is usually more intimate if the hemorrhage be in the upper part of the intestine. The color of the blood in liquid stools sometimes forms a good criterion for determining the seat of hemorrhage. The higher the hemorrhage is, the more altered is the original blood color, due to intestinal putrefaction and intestinal digestion. In hemorrhage from the stomach, gastric digestion may also cause some change. Profuse hemor-

rhage from the stomach does not always cause hematemesis, and we may first suspect the condition from the appearance of the stools, which may be nearly black and of a tar-like consistence. Profuse typhoidal hemorrhages sometimes show an alteration of the blood; usually, however, it is still distinctly red, because it generally comes from the ileum, and a stool follows a hemorrhage quickly, the hemorrhage acting as an intestinal stimulant and in a way like lavage. The bloody, serous diarrheal stools without true fecal matter are of diagnostic importance in certain types of ileus, especially invagination. It may finally be mentioned that the microscopical examination, or better yet, the chemical examination, of the feces for blood is of much significance in connection with carcinoma and ulcer of the stomach.

Pus may be present in the stools mixed with the fecal material. A very considerable admixture of pus in the stool is always due to perforating abscesses. A slight amount of pus in the stool, which often can be demonstrated only microscopically and which is frequently associated with blood or mucus, may be due to catarrhal changes, but is generally due to ulcerative processes of the mucous membrane. Lumps of pus indicate an ulcerative process. It may be very difficult to demonstrate pus in the stool, because the pus-corpuscles are sometimes destroyed by duodenal digestion and putrefaction in the colon. It is a well-known fact that intestinal decomposition destroys pus-corpuscles within a very short time, making it impossible to recognize them. However, even in a normal stool, individual polynuclear leucocytes may be found as a result of the normal transmigration through the mucous membrane. The demonstration of pus is sometimes difficult, even in perforation of perityphlitic abscesses into the intestine, because decomposition in the abscess has already begun to change the character of the pus, this change becoming still greater in the colon.

Tumor fragments may be met with in the stool in cases of carcinoma of the rectum or of the intestine higher up. These fragments attract attention chiefly in a liquid stool by their grayish-red color and solid consistence. Fragments of adenomatous polypi are sometimes found in the stool. They may occur independently or they may be located in the neighborhood of carcinomatous and tuberculous ulcers of the intestines.

In cholelithiasis an attack of biliary colic may be followed by the appearance of gall-stones from time to time in the stools, or they may appear without any previous biliary colic. In searching for them the stool should be thoroughly mixed with water and washed and rubbed

through a sieve, the Boas stool sieve being very convenient for this purpose. To be certain, we should examine the stools during at least fourteen days after the cessation of the attack of the colic. Even a careful and prolonged search may fail to find the concretion in certain cases of gall-stone disease, sometimes because the stone, which is impacted in the neck of the gall-bladder and so caused the colic, has returned to the bladder itself. In other cases the stones which are at fault have become stuck in the ductus choledochus while the patency of the biliary passage has been reëstablished without dislodgment of the stone. Finally, the concretion may be disintegrated in the bowel and so escape detection.

The parasitology of the stools is mentioned in connection with the various subjects according to their clinical entities.

MICROSCOPIC EXAMINATION OF THE STOOLS TO DETERMINE THE UTILIZATION AND SPLITTING OF FATS.

Incomplete utilization of the fats sometimes becomes apparent even in a macroscopic examination of the stool. Stools which contain an abnormal quantity of fat appear light colored, gray, peculiarly glistening. Under the microscope the fat is seen to consist of fat-drops and needle-like crystals. Soaps usually appear as thick, short needles or yellow clumps; free fatty acids, as needles. The addition of osmic acid colors fat microchemically brown to black. Yet this reaction is only characteristic of fats which contain oleic acid; whereas the other fats either remain unstained or require the addition of alcohol to bring out a color. The addition of an alcoholic solution of Sudan red colors all neutral fats.

Normal stools contain a moderate number of fat-needles, which represent the fats that are not easily melted, and are, therefore, less readily absorbed. Fat-drops are not so common except after abundant ingestion of milk or easily melted fats. They usually signify an insufficient utilization of the fats, as does also an abnormal abundance of fat-needles, and is of much value in connection with the diagnosis of pancreatic deficiencies from whatever cause.

The microscopic examination of the stools to determine the utilization of starch is well known. Starch-granules are characterized michrochemically by their blue or violet color after the addition of Lugol's solution. Well preserved starch-granules are rarely found in the normal feces of an adult, but they may occur in the stools of infants improperly fed with starches. An abundance of starch-granules in the stools of adults is pathologic, and is usually a sequence of diarrhea or of gastric hyperacidity. The absence of pancreatic juice

does not seem to cause the appearance of pathologic quantities of starch in the feces, because the starches, along with the other carbohydrates, are utilized very extensively by the intestinal bacteria with formation of acids.

The examination of the feces to determine the utilization of the muscle-fibers of the connective tissue and of the other proteids of foods is one of the most valuable methods of examination. The more thorough the digestion, the less the muscle-fibers appear, and the more are the ends of the fragments rounded off. The fibers are tinted brown in part by their own color and in part by the color of the feces. Microchemically, they are characterized by the appearance of a red color upon heating with Millon's reagent. Undigested muscle-fibers are constantly found in the feces of a person upon a mixed diet. The amount becomes pathologically increased in diarrhea, in fever, and in other disturbances of the digestion.

Large fragments of connective tissue are generally readily recognized even macroscopically by their fibrous structure; this also characterizes them microscopically, as does their pronounced clarification upon the addition of acetic acid. They can be distinguished from mucus shreds by their much firmer consistence, more distinctly fibrous structure, and the disappearance of this structure after the addition of acetic acid, which renders mucus striated.

According to Kuhne's and A. Schmidt's investigations, raw connective tissue is digested by the gastric juice only, whereas cooked connective tissue is also easily dissolved by the pancreatic juice. Hence the amount of connective-tissue that remains in the stools depends upon the amount ingested, upon the degree of boiling or the roasting of meat, and upon the gastric function. Starting from these premises Sahli devised his desmoid test. Schmidt estimates the gastric digestion by determining the amount of connective tissue in the stools after the ingestion of his test diet. He claims that the microscopic appearance of the muscle-fibers in the stool of a patient who has ingested a test-meal of 125 grams of very slightly roasted chopped meat demonstrates some severe damage to the intestinal digestion. On the other hand, the occurrence of microscopically visible fragments of connective tissue is indicative of insufficient gastric digestion, because connective tissue, unless it is cooked to pieces, is dissolved only by peptic digestion. After the employment of both the Desmoid test and the suggestion of Kuhne and Schmidt I am free to confess that I have not received much in the way of practical suggestion in clinical work. Instances in which particularly the Schmidt tests have been of help exist, but are not numerous.

EXAMINATION OF THE FECES FOR ESTIMATING CHRONIC CHANGES OF INTESTINAL DISTURBANCE.

The macroscopic examination forms a very important part of the whole procedure, and this alone is often sufficient to enable the experienced observer to form a judgment. In the first place it determines whether color, consistency and odor correspond with the normal feces.

My experience has been entirely in connection with my test diet and what follows corresponds to that. The normal test-diet stool, after having been ground up very small (smaller than pinheads), shows brown or red-brown points (chaffy remains of the oatmeal-gruel, remains of cocoanibs), the nature of which is ultimately explained by a microscopic examination. Otherwise it is entirely homogeneous.

For a closer macroscopic examination the entire excrement is first stirred up thoroughly with a wooden spatula, and a small amount of about the size of a walnut is transferred to a porcelain mortar (about 12 centimeters in diameter). This is ground up as fine as possible with the addition of, at first, a few cubic centimeters and gradually more distilled water (to about the consistency of sauce). The grinding up must be done carefully so that no coherent particles of excrement remain. The ground-up specimen of excrement is then spread over a flat black plate or a large vessel in as thin a layer as possible and account is taken of all the elements that are still recognizable in it by the naked eye.

For the purpose of further examination, a small quantity of stool is stirred up with a small amount of more water and the same is placed between two pieces of plate glass (microscope slides will do), and the specimen is examined by direct light, with or without a magnifying glass. It will then be seen in the normal test-diet stools that there are small brown or reddish-brown forms, the remains of the oatmeal gruel, and of cocoanibs if this has been taken. Otherwise it is entirely homogeneous, with the exception of here and there a possible flake of cellulose substance which is of a white color. Large areas of light color are strongly suggestive of lumps of mucus.

Under pathological conditions the remains of fibrous tissue and tendons of chopped meat may be seen. These have a whitish-yellow color and a thread-like appearance with a rather solid consistency, which when treated with acetic acid (36 per cent. sol.) may be caused to disappear, but if, on examination under the microscope, this appearance becomes more marked, mucus is present.

Remnants of muscle-tissue, very small brown-colored rods, may be observed. When these are of considerable number they are indicative of disturbance of intestinal digestion.

Potato-remains appear like sago-grain, glassy granules, and may easily be mistaken for flakes of mucus. They are readily discovered by their globular appearance and larger consistency of the fecal mass. When in doubt they should be examined under the microscope when the potato-cells will show the starch granules, easily colored blue or brown with iodine.

Fat-remains are evidenced by the light color and by the clay-like consistency which causes them to project above the level. Mucus, in larger and smaller, even very small flakes may be observed incorporated with the stool. They are noticed when glassy and soft and easily broken up, but not dissolved. Larger shreds, or strips, such as would be observed in cases of chronic colitis are easily diagnosed by the appearance. Even in cases of marked colitis, however, these shreds of mucus may not be observed, the mucus being incorporated with the stool. Sometimes, even, they are not observed by the method of pressing the specimen between two pieces of glass, but only by a microscopical examination, when small hyaline-mucus islands and yellow mucus-granules would be noticed. Sometimes one can see large crystals of ammonio-magnesium phosphate. These are present only in rare instances so far as macroscopic examination is concerned, and they represent really a condition of entero-colitis.

MICROSCOPIC EXAMINATION OF THE FOOD DETRITUS IN STOOLS.

The microscopic examination serves for the most part to complete the macroscopic; by means of the latter alone pathologic conditions are often not disclosed. Ordinarily, as Schmidt advises, three areas of stool are examined. These may be placed upon the same slide, and covered with a cover glass. The first is examined without anything having been added to it; the second is thoroughly stirred with a small drop, 36 per cent. (official) acetic acid, heated for a moment over the flame (until it begins to boil) and then covered with the cover-glass. The third is rubbed up well with a small drop of a strong solution of iodo-potassium iodide (iodine, 1.0; potassium iodide, 2.0; aq. dest., 50.0).

The normal excrement examined in this way gives the following result: In the first preparation without any addition there is seen a detritus consisting of the smallest granules, globules, bacteria, etc. These are isolated fragments of muscle-fibers, yellow in color with

rounded edges, flake-like structures, occasionally with indications of cross striations, scattered smaller and larger yellow salts of calcium, flakes consisting of fatty acid salts of calcium with irregular borders, bright and deep yellow in color, colorless soaps, isolated potato cells, either empty or with remains of a content, which can no longer be differentiated, scanty chaffy remains from the oatmeal-gruel, often recognizable with the naked eye as little dark flakes, and finally, according to circumstances, where cocoa is taken instead of milk, remains of cocoa.

In the second preparation, the fat-remains are melted out. We thus have an entire fat content visible to the naked eye. The larger salts of calcium and soap flakes have disappeared. If the preparation is carefully heated and placed under the microscope while still hot, the small flakes of fatty acid are melted into drops, which congeal very suddenly as the preparation cools again. In the third preparation, which appears brown in color, the potato-remains ultimately assume a slight violet shade (not blue); the blue colored parts (starch granules or microbes) are generally absent.

Pathologic conditions are suggested in the following findings: In the first specimen, fragments of muscle in larger number and better state of preservation than normal; many needles of fatty acid and soap; drops of neutral fat; numerous groups of potato cells; parasite eggs. In the second acetic-acid preparation, massive fatty acid flakes. In the third iodine preparation, blue colored remains of starch granules; blue or violet fungus spores, and yeast cells, which are colored yellow by the iodine.

CHEMICAL EXAMINATION OF THE FECES.

Under normal conditions the reaction of the feces is neutral. It may however, be faintly acid or faintly alkaline, according to the diet. The reaction of the surface of the feces is often different from that of the interior. The reaction may also change upon standing. It should therefore be taken on fresh stools. Pathologically, the reaction may become either strongly acid or strongly alkaline, according to the kind of decomposition processes which are occurring in the intestinal canal. When marked putrefaction is present, the reaction is alkaline; when marked fermentation is present, the reaction is acid. Red and blue litmus paper answers for the testing. Patients upon a marked milk or starch diet, and those in whom there is deficiency in bile, produce acid stools.

The color of the feces of normal individuals is never due to unchanged bile pigments (bilirubin). The latter is transformed into

urobilin in the intestines and is partly reabsorbed and used over again by the organism. Therefore, the presence of bilirubin in the feces always indicates some abnormal function of the intestines, either some disturbance in the absorption or in the chemical process, or else an increased peristalsis. The pigment is easily demonstrated by means of the Gmelin test, which is accomplished by dropping a little fuming nitric acid upon the feces and noting the green, red and violet rings around the drops of acid. The green ring is the most characteristic.

Schmidt recently described a simple method for detecting urobilin in the feces directly without extraction by means of alcohol containing hydrochloric acid or zinc chloride. He adds a little concentrated mercuric chloride solution to a small amount of feces in a porcelain dish. If urobilin be present, the feces will turn red. The reaction can be completed in about a quarter of an hour. The same test can be employed to demonstrate the presence of bilirubin, which will turn green in the presence of the mercuric chloride solution. In the same specimens, therefore, we can recognize, by the contrast of color, the particles which contain bilirubin and those which contain urobilin. While in a general way the value of this reaction is not of great practical significance, it nevertheless is a means of denoting disturbance in the intestinal canal. The demonstration of urobilin in the stools is of clinical importance in deciding whether in any given case of jaundice the common bile-duct is still partially patent, for in complete obstruction urobilin is absent from the feces.

Because of the large quantities of bacteria in feces the examination of this substance for digestive enzymes is of no general value in a clinical way. It may be performed rather easily, but its significance *pro* and *con* is not so readily estimated.

Mucin in feces, is largely present in catarrhal conditions of the intestines, and is usually made note of by its appearance, but may be tested for by mixing the feces with water, adding an equal volume of lime water, in which mucin is soluble, and then adding dilute acetic acid to the filtrate. Cloudiness indicates mucin. As a general rule, however, the test is of not much practical value and one may safely depend upon the microscopic test for the presence of mucin.

The significance of the presence of blood in the feces is very great, in the early diagnosis of cancer of the stomach or of the intestines, ulcers of the stomach, tuberculosis of the intestines, typhoid fever, and other conditions of the digestive tract associated with hemorrhage, such as various simple forms of ulceration, all of which

show the presence of more or less blood in the feces. Sometimes this is invisible even under the microscope, and must be examined for chemically. The best tests to use are the Weber guaiac test with hydrogen dioxide, or what suits my purpose best, the aloin test, in which an acetic acid ether extract is employed. The benzidin test which answers quite perfectly for testing gastric contents is too sensitive for feces work. Of course, the spectroscope may be used, although it is not found in a sufficient number of laboratories to be employed generally.

FERMENTATION TESTS.

Schmidt has recently endeavored to draw conclusions in relation to intestinal function from the manner in which the feces ferment after evacuation, *i.e.*, from the gases which are thus formed. Such fermentation, if it be very marked, depends essentially upon an abnormal content of carbohydrates in the stools. Schmidt's direction for performing his fermentation test is as follows: "5 grams feces are selected from a well-mixed stool by means of a wooden spatula. Much less is taken from a hard than from a watery stool in order that one may work with approximately equal amounts of dry substance. No greater accuracy is essential for diagnostic purposes. The material is well stirred with water, put into the bottom glass (*a*) of the fermentation apparatus, which is corked with a rubber stopper, taking care to prevent the entrance of air bubbles. Tube (*b*) is filled with tap water and closed with a small rubber stopper, the same precautions against admission of air bubbles being taken. The apparatus is then assembled as in the figure, and left in an incubator at 37° C. for twenty-four hours. Tube (*c*) has a tiny hole at the top and should contain no water. If gas develops from the feces, the corresponding amount of water will be driven into tube (*c*) and its height can be read off." Schmidt considers the test positive (*i.e.* indicating an insufficient utilization of the carbohydrates) when about 1 gram of dry fecal substance passed after a test diet produces more than one-quarter tube of gas. The height of water in the ascension pipe is noted; vessel (*a*) is opened and the reaction of it may be tested with litmus paper, and compared with the reaction before the test was instituted.

Normally no gas, or little, is formed in the incubator test, and the original reaction of the feces undergoes no important change. If gas is developed to such an amount that one-half or more of the ascension pipe is filled with water, then pathologic conditions are present, and if at the same time the reaction has become distinctly

more acid, carbohydrate fermentation, the positive result of the fermentation-test, has occurred; if it has become distinctly more alkaline, albumin putrefaction has occurred.

In the presence of fermentation the opened glass (*a*) gives off the odor of butyric acid, and in the presence of putrefaction, a decided putrefactive odor.

In a general way it may be said that putrefactive stools are

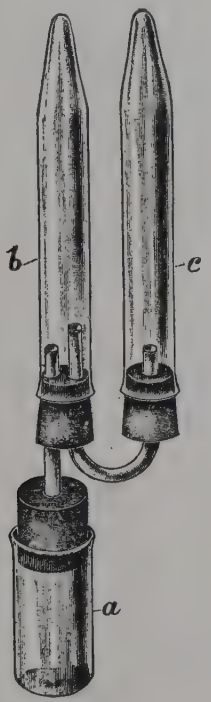


Fig. 42.—Strassberger fermentation tube apparatus.

dark colored and hard, while those which are mushy, containing numerous gas bubbles and smelling strongly of acid, are due to fermentation, usually in the colon.

Because of the fact that more can be learned by a more careful examination of the feces than is possible by the Schmidt fermentation test, several years ago I devised a modification of it, and this modified method, which at the time was described by me, I have had no reason to change my opinion about up to the present. The method which follows represents the routine in my laboratory work instead of the more crude Schmidt test which has been described:

The tests are made by means of an especially constructed fermentation tube, the upright closed or anaërobic limb of which has a 15 cubic centimeters capacity graduated in per cents., and an open bulb of 30 cubic centimeter capacity. The apparatus over all is less than 6 inches in height, will go into a medium-sized incubator, and is constructed so that 25 cubic centimeters sufficiently fills the instrument. For the chemical test of feces 7.5 grams of the soft end of a stool (mushy consistence) or 10 cubic centimeters of a more liquid stool is taken, each of which is mixed in 25 cubic centimeters of sterile water; in the bacteriological tests with media, the tube is filled with a 2 per cent. dextrose-bouillon and inoculated with about three or four drops of a watery suspension of feces or a little less amount of the feces itself. In the case of the chemical tests of the feces, the weighed portion of the stool is thoroughly mixed with water in a casserole by means of an ordinary teaspoon and any very large sticks of cellulose or vegetable substance removed, more feces being added to make up the shortage. The fermentation tube (*a*) is then placed in the water jacketed oven, where it remains at 37° C. for twenty-four hours, when the examinations are made. If at the time of examination more or less floating feces and incorporated gas are seen at the top of the fluid in the upright limb, several fair-sized shot are placed into the bulb, which is then filled with water, corked so that no air from without is present between the fluid and the cork, and the instrument is shaken so that the shot splits up the floating feces, after which the gases that have escaped into the bulb are passed back into the closed limb, the cork is withdrawn, some of the fluid is run out, and the observations made as follows:

First, the total gas content is observed, after which in the chemical or bacteriological feces tests some of the fluid is withdrawn from the bulb and examined for indol and urorosein, and with the nutritive media also for aërobic bacteria, care being taken that the tube is not shaken and that the one or two drops of media taken be from the film on the surface and at different levels under this but not actually down to the crotch between the two sides. The next step in the nutritive media test is to obtain samples of the important anaërobic bacteria. These are best extracted by means of a pipette (*b*) which should be carefully introduced into the lower part of the anaërobic limb and about 0.05 cubic centimeter of media sucked up. In tests to note the amounts of bacteria growing at different levels of the fluid media in the closed limb I have found that at the end of twenty-four hours most of the anaërobic bacteria are at the bottom of the tube, although they also are scattered throughout the closed

limb and thus may be obtained from the upper layer of fluid when the cock has been opened. The carbon dioxide in the accumulated gases is now absorbed by introducing into the closed limb with the pipette 2 cubic centimeters of a saturated solution of sodium hydrate (making sodium carbonate) and the tube is inclined to permit this heavy solution to run some distance up into the closed portion and

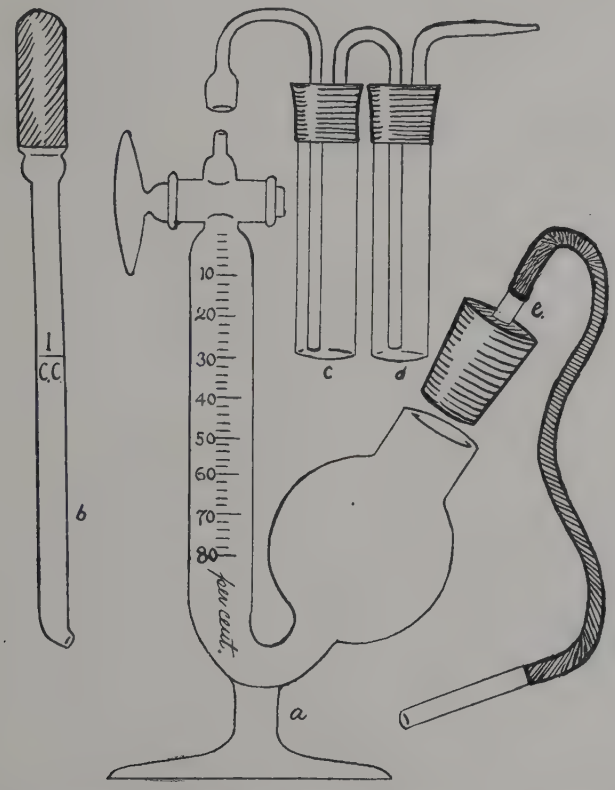


Fig. 43.—Author's gasometer fermentation apparatus for raw stool, and the bacteriological examination of same.

by allowing it to stand for the time necessary to observe that the fluid no longer rises in the tube. In a number of tests to note the accuracy of this chemical method, I have observed that the remaining Co_2 is never more than from 5 to 10 per cent. of the original amount, and this is close enough for clinical work. The remaining mixed gases are then tested in the twin bottles, they being driven through when the cock is opened by means of attachment (*e*) and blowing. The reactions of the gases are noted in the first bottle (*c*) which

contains a weak solution of neutral azolitmin (which is a pure litmus about 200 times as sensitive as commercial litmus), a control in a test tube being used to note the color change. In addition to the reaction of the gases, in the chemical and bacteriological feces tests, the fluid in the apparatus is tested at the end of the twenty-four hours to note the chemical change. The second bottle (*d*) contains lead water (sol. plumbic acetate 10 per cent.) to note the presence of sulphurated hydrogen (turns darker or black according to amount present.) The hydrogen and marsh gas may be burned at the drawn tube outlet, but as both burn with an indistinguishable blue flame and as neither of these gases are important in clinical ways, this may be omitted.

To arrive at some standard of gas result from watery suspension of feces I have made observations of feces with my method from fifty apparently normal persons, and comparing these with the many tests of feces from abnormal individuals I have come to the following conclusions: The Schmidt and Strassburger test diets (first No. 2 and then No. 1) do not give better results than that which I have suggested. For this reason, I have discontinued the use of the difficult to institute and carry out, Schmidt and Strassburger diets, and have employed the diet described. The Schmidt diagnosis of "fermentative dyspepsia" is a most vague and impractical one for clinical work and means but little to base our therapeutics upon. The gas results from the diets I have suggested may be stated as from 10 to 30 per cent. for the ordinary mixed diet; less than that, from 5 to 20 per cent., for a meat and water diet; and more for a carbohydrate and water diet, namely from 15 to 40 per cent. On a strict meat and water diet it may fall to one-half, and on a strict carbohydrate and water diet it is usually higher. The reaction of the fluid should always remain neutral, never becoming definitely acid or alkaline. With the use of the nutritive media inoculated with feces, from 20 to 30 per cent. is noted, never below or above. With the bacteriological tests, special diets are not important and in fact are not advisable. Other points are that the reaction of the gases should never become definitely acid or alkaline, or perceptibly large amounts of indol or urorosein be present in the fluid at the end examination.

Local disorders of digestion in the intestines, in my opinion, make up far more cases of affections of the digestive canal than those due only to local disorders in the stomach. In his work with Krehl, Cohnheim studied the problem experimentally, and found, significantly that it was almost impossible to produce functional derangements of the stomach by direct injuries to the organ itself.

Using dogs which had been provided with both gastric and intestinal fistulæ, these investigators showed that injury to the intestine affected not only the functions of the intestines, but also to a marked degree those of the stomach. They were able to prove that following an injection of magnesium sulphate (Epsom salts) into the intestine the amount of stomach secretion at the succeeding meal was increased to nearly double, while after a similar injection of a strong solution of sodium chloride (table salt) the amount produced by the meal was less than one-half. Analogous changes were produced in the total acidity and in the length of time required to empty the stomach. As judged from any observations of feces tests and treatment for these conditions in cases of chronic intestinal putrefactions, a constantly low running degree of toxemia from the bowel can be shown to have etiological bearing upon many of the cases of disturbed sensation, secretion and motility of the stomach, and, contrary to what our sense impressions have taught us, we must seek the origin of many gastric disturbances in the intestine, rather than consider them as only gastric affections or neurotic ones referred from parts other than the digestive canal. In the discussion of a paper on indicanuria at the annual meeting of the American Medical Association in 1909, I pointed out that, contrary to the general teaching, a marked type of indolic putrefaction may and often does exist with a high stomach secretion and motility. In my records then of 127 cases of indolic putrefaction, 79 showed a hyper, or normal acid secretion, and in the 48 in which this was below normal or absent, 34 subsequently gave evidence of improvement in the stomach secretions when the bowel conditions became improved. Thus in gastric work the feces test for bacteria should never be omitted. Indicanuric conditions (which as a clinical entity is no more significant to me than edema or cough would be), must be looked upon both as an aggravator and depressor of stomach secretion, and the stomach conditions mentioned are not to be considered as neurotic in nature unless the bacteriological feces examinations are negative. For the basis of these intestinal conditions the most useful clinical classification is that of Herter, namely, the indolic, saccharo-butyric, and the combined forms. Considering that from the bowel a normal individual voids about 130 billions of bacteria a day of many different types and that the performing of culture methods for segregation are almost impossible for clinical work, the apparatus method answers well and to additional clinical purpose for diagnosis than only a simple gram differential stain of the raw feces. Fortunately, there are three main anaërobes prominently found in all of these cases,

and each of these grow well in the closed limb and on the media mentioned. They are the Bacillus coli communis, to note the indolic form, and the Bacillus aërogenes capsulatus and the Gram-positive diplococci to note the saccharo-butyric form, and thus when any of these bacteria grow in wild and predominant proliferation in the media the diagnosis and therapeutics in the case are suggested.

GENERAL REMARKS AND NORMAL STANDARDS.

In healthy man, the natural secretions of the intestine with the defecations hold the bacterial content down. Under ordinary conditions there are few colon bacilli in normal feces. Most of the bacteria are present in the colon, where the anaërobic conditions for their proliferation are perfect. The putrefactive processes are due mostly to the anaërobes. As a rule, a patient on a meat diet, harbors fewer organisms, and thus less gas, than those on a mixed or carbohydrate diet, but this need only be considered in a relative way in the bacterial tests. Indol from putrefaction of the proteids is not due to tryptic digestion, but to bacteria, and mostly to the bacillus of the coli group and not the Bacillus aërogenes capsulatus or G. P. diplococci. As the liver and renal cells, if normal, produce enough oxydase to bind the indol but very small amounts of indol may appear in the urine. But if the supply of indol be great, or the liver and kidney function be deficient, larger amounts of indol are supplied to these organs and, being unbound, appear in the urine, and then the irritating effects on these organs, as well as other structures in the body, are produced.

Human bacillus coli communis have practically no power to dissolve and peptonize native proteins, but when these are first affected by the other organisms, the colon bacilli are able to energetically cleave the peptones and give rise to ammonia, volatile, fatty acids, phenol, indol and hydrogen sulphide. The Bacillus aërogenes capsulatus is a most abundant gas-making organism in dextrose bouillon cultures and generates hydrogen, carbon dioxide and butyric acid. Therefore, because of the mixed association of these and other influencing bacteria in the gut, the gas result from nutritive media in general must not be considered as too clinically distinctive. Nevertheless, paradoxical as it seems, in the true cases of the saccharobutyric type of chronic excessive intestinal putrefaction the gas result is generally low, usually only from 5 to 13 per cent., whereas in the indolic form this may run as high as 80 per cent. The normal standard I employ is that of gas from 20 to 30 per cent. It should

be recalled that normally with the feces and water suspension, a meat diet gives a lower gas than the ordinary diet, and a vegetable higher than the ordinary, while in the states of chronic excessive intestinal putrefaction, the indolic type (due mostly to bacteria protein cleavers) gives a higher than normal, and in the saccharo-butyric type (mostly bacteria carbohydrate cleavers) a lower than normal. Therefore, a patient whose feces gives a higher than normal gas result in nutritive media should be placed upon a strict meat diet and the watery suspension feces test performed, and if in this the gas is higher than the meat diet should normally be, the diagnosis of the indolic form is suggested; and to reverse this rule, when the gas result in the nutritive media is less than normal, the patient should be placed upon a strict carbohydrate diet to see if the gas from watery suspension of feces still remains below what is ordinarily seen in an individual on a strict carbohydrate diet, in which case, when positive, the diagnosis of the saccharo-butyric form is suggested.

But to arrive at these diagnoses more conclusively the urine must also be examined for the different substances, and also the bacteria grown in the nutritive dextrose bouillon stained and examined. In the indolic and combined form, indol in large amounts is usually present in the urine; in the combined to a less extent, but none at all in the true saccharo-butyric form, in which urorosein (Jaffé test), large amounts of oxaluric acid crystals and phosphates are more commonly seen. With the Gram differential staining method the bacilli of coli group, being Gram-negative, are stained with the counter stain (red with carbol-fuchsin), and the bacillus aërogenes capsulatus and diplococci, both Gram-positive, staining violet with the gentian violet, the distinction of colors serving well to differentiate them. The organisms of the colon group are very small, the bacillus aërogenes capsulatus very large, like thick rods, and the Gram-negative diplococci are also quite large and easily distinguished. In both types of putrefaction these organisms are found in the closed limb of the apparatus and easily observed.

BACTERIA OF THE FECES.

Feces consists to a large extent of the body substance of micro-organisms. There are many different varieties found. Of late the bacteria and fungus of normal intestinal content have been extensively studied. The majority of organisms belong to the colon group. The examination of the stools for micro-organisms furnishes information concerning bacteria in the colon. The contents of the small intestine, such as is obtained from fistulæ are generally different.

Not only is the small intestine much poorer in bacteria, but it also contains different species, especially in the liquefying bacteria. Barely 1 per cent. of all the bacteria passed in stools is viable by ordinary cultural methods. This does not assume that they are all dead, but to some extent that the type of media used is not suitable for their growth. Bacteria comprises about one-third of the dry residue of the feces. Strassburger estimated that in twenty-four hours a healthy adult evacuates about 8 grams of bacteria, dry weight, and that in diarrhea the bacteria may increase from 14 up to 20 grams. In constipation there is a decrease to about 5 grams. Reduction of food lessens the daily quantity of bacteria.

The quantitative determination of the bacterial content of feces possesses value in clinical work. This is accomplished by taking a weighed portion of feces, thoroughly mixing it in water, centrifuging it when the heavy particles of feces will be separated from the fluid. The watery remains are then mixed with double the quantity of alcohol, recentrifuged, dried and weighed. From the quantity of bacteria found in a cubic unit of stool calculation of the daily output may be made, or the quota of bacteria to the food residue estimated.

According to Schmidt and Strassburger the examination of the stool for bacteria possesses little significance in a clinical way, because the character of the bacteria can be more or less modified and controlled according to the character and the quantity of the diet. They quite discourage the determination of the bacteria as of any clinical significance, and the same is true with quite a number of observers since their time. To me, however, the examination of the stool for bacteria possesses the most valuable means that we have today to diagnose various types of chronic intestinal fermentations and putrefactions, as well also as states of infections of a chronic nature connected with what is known on the subject of toxemia and also various ulcerative conditions of the colon and rectum and congested catarrhal conditions. It is far from a simple method of procedure, and that is the unfortunate part of it. Not only is a well equipped laboratory and much time necessary to do this work, but one must be skilled in knowledge and understanding of bacteriology, in the technique of growth and examination, and in the ability to interpret the findings. It is now some years ago that I engaged intensively in the study of intestinal bacteriology, and I have not regretted a moment that I have spent upon it. Definitely, I feel convinced that much of the benefit accruing to the patients I have had has come from the findings in the laboratory in the bacteriological study of the feces. What is mentioned in the following pages possesses particular

significance in connection with the diagnosis and treatment of many chronic intestinal disorders.

If one examines with the microscope the contents of any portion of the intestinal canal of a mammal, the richness of the material in micro-organisms is strikingly apparent, especially in the stained preparations. Their number has been estimated at about one hundred and thirty billion for the daily human excreta. As far as the feces are concerned many of the organisms are no longer living and are undergoing a process of disintegration, partly owing to a solution in their own juices—a process of autolysis. The bacterial inhabitants are numerous and they represent many species and varieties. The studies of Nuttall and Thierfelder[2], and those of Levin[3], tend to show that the bacteria in the human intestinal canal are not essential to life and normal nutrition. Even with a sterile intestinal canal the mammal will live and be normal in nutrition. The polar bear has essentially a sterile intestinal canal during life, providing the excreta from him is examined while he is still in the Arctic region. When he comes into captivity in the various zoos, etc., the intestinal canal soon manifests the presence of bacteria and in a short course of time the number of bacteria are essentially as in all of the domestic animals. The real significance of the normal intestinal flora probably lies not in any immediate relation to processes of digestion, but in a wholly different direction. These bacteria undoubtedly gain entrance in the human being with food and drink, and if they find conditions suitable to multiply in the digestive tract, this is accomplished. Many foods, such as water, vegetables, salads, milk, cheese, oysters, game, etc., are liable to contain injurious bacteria. Among such are the pyogenic streptococci and staphylococci, the paratyphoid bacilli, typhoid bacilli, the dysentery bacilli, proteus vulgaris, spore-producing anaërobes, etc.

The normal human being is provided with more or less efficient methods of defense against these bacterial invaders. This is accomplished mainly by the secretion of the gastric juice, which provides a degree of acidity which acts as an effective check upon the growth of many non-sporulating bacteria, and is actually destructive to most varieties, at least in some measure. In many others, however, bacteria are present in the stomach due to interference with the motility and secretion, and many people over forty years of age and in fair health have had their digestion sufficiently broken down to be significant. The bacteria then find their way to the region of the colon and here are confronted with immense numbers of the chief obligate race of bacteria of the digestive tract, the main representatives of which are those of the B. coli type. Another group of obligate organisms,

closely allied to B. coli, the B. lactis aërogenes, is present in the upper part of the small intestine and becomes gradually less abundant with its descent into the colon and finally appears in relatively small numbers in the feces. However, while this latter type, the B. lactis aërogenes, gradually diminishes as it descends into the level of the gut, representatives of the B. coli group grow more and more abundant and beyond the ileocecal valve they largely dominate the intestinal flora.

It is a well known fact that between the members of the B. coli group, characterized by a certain hardiness in growth on ordinary media, and the free production of gas and acid on various sugars, by the coagulation of milk, and usually by the formation of indol, there exists distinct antagonism with various others of the intestinal flora. Harden[4] has shown that there is a constant difference in the behavior of B. lactis aërogenes and B. coli when grown anaërobically on sugar bouillon. There is a distinct, far-reaching contention relating to the defensive action of the B. coli group based upon the observation of Conradi and Kurpjuweit[5] that the members of this class make thermostabile and thermolabile substances which have a powerful antibacterial action, being still active in a dilution of 1 to 10,000 parts, and hence comparable to the antibacterial action of carbolic acid. The inhibitory action of these substances is stated to be not confined to alien bacteria, but relates also to the B. coli group. Inefficiency in growth observed in all cultures was attributed to this substance. Recent studies of this bacteria strongly suggest it to be a protective inhabitant in the digestive tract. The acidophile bacteria, for instance, assumes a dominant position in the digestive tract if provided with suitable food materials for the production of acid, and it is a fact easily proved that when the acidophile bacteria are large in numbers the quantity of the more serious types are in the minority. Various facts that are easy to demonstrate in the laboratory serve to prove that micro-organisms of the B. coli type are able under some conditions to check the growth of pathogenic micro-organisms which are also often found among the intestinal flora. Thus it is that these bacilli are essential to the life of the mammal as a defense against bacterial foes which it is impracticable to wholly exclude from the digestive tract, and not as agents in directly facilitating the processes of digestion in the narrow sense. My observation suggests that only four of the now known seventeen different varieties of B. coli communis group are pathogenic in type, and that the rest of them act as defenses against the activity of the more serious types of organisms. There are many examples of disease in which the colon bacilli dis-

appears, not only from the feces, but from the diarrheal stools, other races taking their place. This condition is quite unintelligible unless we assume that the colon bacilli no longer finds a suitable nutrient medium in the mixture of digestive juices and food. Their return in many cases would seem to show a return of the more normal secretory condition in the digestive canal as an influencing factor in determining the reinstatement of the legitimate flora.

The intestinal canal of man is essentially anaërobic, particularly so in the small intestine and somewhat less so in the colon. There is a big change in the bacteriology noted in early life compared with that noted in adult life, and even from adult life with that of the

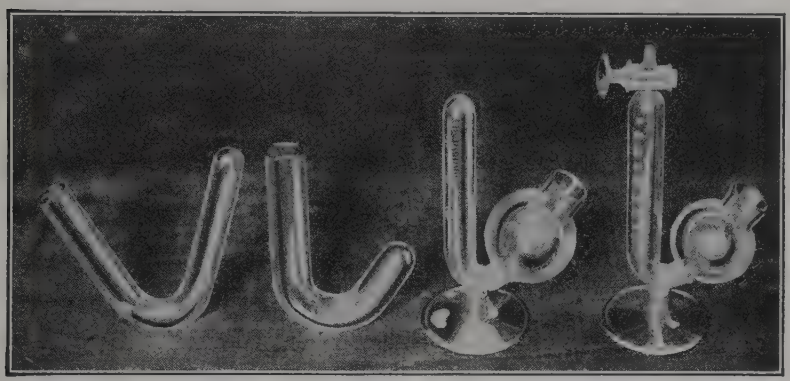


Fig. 44.—Various forms of fermentation tubes used by the author. The full capacity of the anaërobic limb of each of the forms is 15 cubic centimeters. From left to right, the first form is inexpensive and a measure may be used to quickly compute the per cent. of gas. The second is a shorter and wider calibered form with a higher aërobic side so as to allow displacement without the fluid running out of the instrument. The third, an ordinary saccharometer tube graduated in per cents. The fourth, a larger saccharometer with ground glass cock at top. This latter form is the one used in bacteriological and feces fermentation and putrefaction work wherein the gases are estimated, and also has an improvement over the third in that it can be more thoroughly cleaned.

period of senescence. The bacteriology in early life is essentially fickle as compared to that found in the adult. In adult life the bacteriology multiplies, there being many new forms of organisms which gain entrance into the canal and finally live at the expense of the host. Now the presence of the B. aërogenes capsulatus can be easily determined. Later on in life the organisms of the Gram-negative form take precedence, the main ones being those of the B. coli group, and the putrefactive anaërobes as noted in adult life become relatively fewer in number.

It is a well known fact that the epithelial cells of the digestive tract have to do with the protection of the body from the invasion of bacteria within the tract. There exist many reasons, however, to prove that bacteria may pass through the mucous membrane and gain entrance to the peritoneal surface. Numberless experiments have been made to prove that this migration of bacteria within the contents of the gut is a common state of affairs.

The aërobic and anaërobic characteristics of micro-organisms are of great importance in the classification of intestinal bacteria since the putrefactive processes in the digestive tract are carried on largely through the agency of strict anaërobes. No study of the bacteria of the gastro-enteric tract can be considered thorough which does not take the strict anaërobes into account. This, of course, involves the use of anaërobic technique.

By far the most helpful method of studying the microscopical fields is with the aid of the Gram stain. The routine examination of the intestinal contents by means of this stain is a real aid in forming a judgment as regards the presence or absence of certain types of bacteria in the digestive tract. However it is not sufficient as there are instances in which Gram-stained microscopical fields appear entirely normal, but after cultural and chemical studies it is evident that abnormal decompositions are at work and that pathological micro-organisms are present. Nevertheless one may often form a practical opinion from the mere study of the fields as to whether the bacteria present are capable of initiating putrefactive processes or not. I would advise in making these slides to use the very smallest possible quantity of feces and to make a uniform smear. Also that the smear should be as thin as possible; an addition in doing this work are the cultural methods which are most important, particularly after a purgation when one obtains more viable organisms than stool collected under normal conditions. In this connection I will also add the method I suggested—namely, using extract of feces collected from normal individuals which have been sterilized by passage through a Bergfeld or Cumberland filter, the fluid obtained being used as a media without boiling or sterilization.

In addition to the organisms mentioned below, various forms of saprophytes are also important. The following is a list of the organisms which can be recovered from the human intestinal canal.

Bacillus acidolacticus
Bacillus acidophilus
Bacillus aërogenes capsulatus
Bacillus adhesioformans
Bacillus alkaligenes
Bacillus anthrax
Bacillus anthrax symptomatici
Bacillus bifidus

Bacillus botulinus
Bacillus butyricus
Bacillus cloacæ
Bacillus coli communis (17 varieties)
Bacillus dysenteric
Bacillus entericus
Bacillus enteritides sporogenes
Bacillus fecalis alcaligenes
Bacillus flavosepticum
Bacillus flexneri
Bacillus fluorescens
Bacillus fluorescens non liquefaciens
Bacillus Friedländer
Bacillus Kruse
Bacillus lactis aërogenes
Bacillus liquefaciens ilei
Bacillus mascerans
Bacillus odematis maligni
Bacillus paralacticus
Bacillus paraputrificus
Bacillus paratyphoid
Bacillus perfringens
Bacillus prodigiosus
Bacillus proteus vulgaris
Bacillus putrificus
Bacillus pyocyaneus
Bacillus Shiga-Kruse
Bacillus subtilis
Bacillus typhi
Bacillus violarius acetonicus
Single cocci
Double cocci
Staphylococcus intestinalis
Staphylococcus pyogenes
Staphylococcus pyogenes albus
Staphylococcus pyogenes aureus
Streptococcus lacticus
Streptococcus pyogenes
Saprophytes { Gram negative / Gram positive }

The influence of diet must not be overlooked in connection with the bacteriology present in the intestinal canal. It must not be believed, however, that the bacteriology can be influenced definitely and permanently in desirable directions by means of diet alone.

Attention should now be drawn to bacteria in cases of disease. Those which are capable of producing clinical conditions may be enumerated as follows:

The colon bacilli
Typhoid bacilli
Paratyphoid bacilli (*a* and *b*)
Dysentery bacilli
Flexner and Shiga
B. bulgaris
B. proteous
Streptococci and staphylococci
B. bifidus
B. putrificus
B. aërogenes capsulatus
B. botulinus
B. butyricus
B. adhesioformans
B. pyocyaneus
G. P. cocci
G. P. diplococci

to which may be added the various forms of saprophytes, particularly those of the Gram positive forms.

The different types of infections in connection with the various types of disease and toxemia will be taken up in connection with those subjects. It suffices here to add that the bacteriology of stools requires considerable experience and quite intensive study. The average bacteriologist possesses little knowledge of methods of identification and the significance connected with the different types of infection, particularly those of the chronic forms of the human intes-

tinal canal. The same may be said of many clinicians, and even those who are engaged in gastro-enterological work. Some even have gone so far as to state that the bacteriology of the intestinal canal possesses no significance in connection with gastro-enterological disorders. The answer to that is that if such individuals engaged extensively in the study of the bacteriology of the human intestinal canal they would come to a different conclusion. Some rather noted bacteriologists also have made this statement, and a study of their writings suggests conclusively that their statements are made because they know too little about this line of work. It is not sufficient for a person to state that there is no significance to a subject simply because he is not acquainted with it. Arguments based upon the understanding of the subject and also upon the individual research work are necessary before statements such as these can be taken as valuable.

THE EXAMINATION OF THE URINE IN INTESTINAL DISORDERS.

It would be quite out of order in a work like this to include all that pertains to the clinical examination of the urine. Only such things will be mentioned as belong directly to the subject, and then if further information is desired the reader is advised to refer to a book on clinical diagnosis.

The daily volume of urine excreted by a healthy adult varies under average condition between 1500 and 2000 cubic centimeters. While the total amount of urine is dependent upon the ingestion of food or fluid, it is a common clinical finding in digestive disorders to have a scanty excretion of urine, that is, one that is only about one-half in quantity, or perhaps two-thirds. Such a urine, of course, would be higher in specific gravity, of high color, and represents a urine such as one would be accustomed to see in continued fever.

In jaundice the urine contains bile pigments, and its color varies from the dark yellow or green to a brown or black depending upon the concentration of the urine and the amount of bile-pigments or upon its chemical modification. Such a urine is generally strongly acid, and if allowed to get cold, needle-like crystals of urobilin separate and settle with the rest of the sediment. A large amount of urobilin in the urine generally shows a dark-brownish color. Various aromatic products, such as result from decomposition by putrefactive processes are often responsible for a remarkably dark-colored urine. Sometimes this color is not observed until after the urine has stood for a while, it becoming oxygenated.

The normal urine has an acid reaction. It is not uncommon to find in abnormal conditions of gastric and enteric digestion that the acidity becomes diminished.

More or less amounts of albumin in the urine are not uncommon in cases of enteric disturbance, particularly those of an intensive nature. Very often traces of albumin are met with in cases of chronic intestinal putrefaction, and sometimes in fermentation, the albumin disappearing when the intestinal condition has improved. This may be classified as a dietetic form of albuminuria. Not uncommonly there is an output of a large amount of phosphates in which a small amount of albumin may be present.

So-called albumosuria is not an uncommon finding in urines of gastric and intestinal cases. Such albumose urines are found in acute yellow atrophy of the liver, phosphorus poisoning, in ulcerations of the stomach and intestines, where the condition is known as enterogenic albumosuria, in most febrile conditions, especially in infectious diseases, and particularly in the suppurating processes. The presence of the albumoses in the urine is only of limited diagnostic value, but when it is very pronounced and when there are no other reasons for albumosuria, it may lead to the recognition of suppuration. Its symptom may be considered in connection with the diagnosis of perisplenitis, the differential diagnosis between tubercular and peritoneal conditions and in peridiverticular abscesses.

The biliary pigments of bilirubin and biliverdin, the latter being derived from the former by oxidation in the spontaneous decomposition of bile by putrefaction, and perhaps by the action of oxydases, may be present. Whenever these substances get into the blood, they appear in the urine; as in jaundice. Such urines are recognized by their color, or by the Gmelin test, or by the Trosseau test with iodine. In cases of obstructive, retentive jaundice, there may be bile acid present, the Hoppe-Seyler test being sufficient to recognize its presence.

Important in this connection is the presence and detection of phenol. Traces of phenol in the form of phenyl sulphuric and phenol glycuronic acids are normally present in the urine. Pathologically, phenol increases in the urine with increased intestinal putrefaction, and in pus-formation there is almost invariably quite a large amount. The presence of ferric chloride will produce a violet-blue color in the distillate from a phenol urine to which 5 per cent sulphuric acid has been added. The presence of red indol and skatol pigments are detected by the methods described by Rosenbach, and seems to depend principally upon the formation of indigo red, an oxidation

product of indoxyl or indican. It may depend, too, upon the formation of skatol pigments by means of the oxidizing influence of nitric acid. The test therefore is performed by adding concentrated nitric acid drop by drop to a test tube of urine while it is being continually boiled. The urine gradually assumes a Burgundy-reddish color, while the foam produced by shaking turns a bluish red. In my experience it is quite as easily detected by means of the indican test which will be described in which the red indol can be separated from the supernatant fluid by the chloroform, producing a color somewhat allied to the iodine test, and at the same time it will be noticed that the supernatant fluid is distinctly a reddish-purple color.

Urorosein is the name given to a rose-pink color noticed in urine upon the addition of mineral acids, especially hydrochloric. The substance is not soluble in chloroform, and therefore can be noted in the indican test described. It differs from indigo-red and may possibly be a derivative of skatol. Its color can be made to disappear upon the addition of alkaline carbonates to the urine.

Urobilin is a derivative of bilirubin. It occurs in small amounts in normal urine, but has clinical significance when it is of easily detectable quantity. It is found increased in many cases of jaundice and liver conditions. It also is present in certain fevers, hemorrhage and blood disorders. To demonstrate its presence in the urine it must first be extracted by gently shaking a specimen of urine, acidulated with a few drops of hydrochloric acid, with one-third its volume of amyl alcohol. When the alcohol has extracted the urobilin, it will be colored brown. Several drops of an alcoholic ammonium chloride solution and some 1 per cent. alcoholic solution of zinc chloride are then added to the amyl alcohol layer, and the resulting fluorescence shows the presence of urobilin. In my opinion urobilin is a definite chemical substance, and a valuable routine test.

The significance of the presence of grape-sugar in urine and the tests will not be described here. Just a word may be added that they have some significance in connection with a few acute and chronic pancreatic cases. The Cammidge reaction which was offered as an easy diagnostic method for disease of the pancreas is only to be mentioned to be discouraged in clinical work.

Occasionally one meets with the presence of acetone or oxybutyric acid in the urine. Acetone is not infrequently seen in instances of starvation, inanition, in those upon a purely meat diet and in some cases of carcinoma. Its significance in diabetes mellitus is well known.

The presence of leucin and tyrosin are rarely met with but their presence is very characteristic of acute yellow atrophy of the liver, phosphorus poisoning, and may occasionally be seen in fevers, particularly of serious grades. After proper separation they are easily recognizable by their shape of crystals by means of the microscope.

The presence of uric-acid crystals is of significance in connection with some cases of saccharo-butyric fermentation; a common accompaniment, and probably the causative factor in the production of what is clinically known as gout. Great quantities of uric acid are sometimes met with in urines of people suffering from chronic forms of gastro-enteric conditions.

The excretion of phosphates in urine is often of importance, particularly in connection with metabolic disorders and digestive disturbance. Especially is this true when there has been considerable effect upon the general nervous system in which there seems to be as a clinical symptom an increased output of phosphorus in the urine. The uranium nitrate solution method answers best for the purpose of estimation.

Of much significance in clinical work is the estimation of sulphuric acid and the combined sulphuric acid in urine. By the combined sulphuric acid is meant the sulphuric acid united with organic substances, such as phenol, indol, etc., to form the so-called ethereal sulphates, which are of the greatest interest. The best test for the estimation of total sulphur, total sulphate, ethereal sulphur and neutral sulphur is that of Folin. A simple test has been suggested by Emerson, and consists of using a large test tube holding about 25 cubic centimeters. Three volumes of urine are mixed with one volume of an acid chloride solution containing four parts of barium chloride, one part of hydrochloric acid and sixteen parts of water. The sulphates are precipitated as barium sulphates. If a milky opalescence takes place, the sulphates may be considered normal; if the precipitate is denser, the sulphates are increased; with a faint opalescence, fainter than normal, the sulphates are diminished. With a normal amount of sulphate the precipitate occupies about one-half the concave portion of the test tube. If the precipitate be filtered off and hydrochloric acid added to the filtrate and the whole boiled, further precipitate takes place due to the ethereal sulphate.

In the Folin test the difference obtained between the result of estimating total sulphate and for inorganic sulphate gives the amount of ethereal sulphate present in the urine. The normal amount daily of total sulphate in the adult is 1.5 to 3 grams of SO_3, and parallels the quantity of protein decomposition. The normal ratio of the daily

amount of the total sulphates to the daily quantity of urea is about 1 : 5, while the ratio of the quantity of the ethereal sulphates to that of the remaining sulphates is about 1 : 10. In many cases of intestinal disturbance of a chronic nature, particularly those represented in the toxic group, the ethereal sulphates are increased and may to the preformed sulphates present a ratio as high as 4 : 10. It has been said that the experiments that have been made upon the estimation of ethereal sulphates and their significance in connection with putrefactive disturbances have not been conclusive. In my experience this is not so. While the quantity of ethereal sulphates may depend upon putrefactive processes in other parts of the body, such as the breaking down of tissues, the presence of pus, etc., if such can be excluded, a large amount of ethereal sulphate is of much significance in connection with the diagnosis of toxemia.

Indican, or indoxyl sulphuric acid, is a product of putrefaction of protein, and is a derivative of indoxyl, which in turn is an oxidation product of indol. While its decomposition occurs in the intestine normally, it is commonly very much increased in the presence of digestive disturbances, particularly when there is a diminished peristalsis. An increased amount of indican in the urine is of some diagnostic importance in helping to locate the seat of an intestinal obstruction. Experience shows that an obstruction in the small intestine usually is quickly followed by a marked increase in the amount of indican in the urine, as contrasted with an obstruction in the large bowel, when there is very rarely any such increase, until perhaps in the later stages. This, while a general rule, has exceptions under clinical observation. It may be generally accepted that in the majority of instances when indican is met with in the urine, the indican is produced in the small intestine, and not in the large, because it is probable that trypsin of the pancreatic juice which favors decomposition and the formation of indican (which is absorbed before the middle of the large intestine is reached) has to do with its production. Constipation only causes indicanuria when putrefactive processes take place. Indicanuria is not found in simple chronic constipation. In this condition resorption is usually above normal.

Leaving out of consideration certain tubercular conditions and those in which the break-down of tissue (such as resorption of pus, which would produce increased amounts of indican in the urine), the significance of this body in connection with chronic intestinal toxemia has a great importance. If the duct of the pancreas be included, the amount of indican in the urine will be diminished. The same is true of deficient pancreatic function. It is a clinical point of

much significance in connection with the diagnosis of pancreatic disease, that commonly marked disease has indican-free urines. It is usually necessary, however, to put these patients upon a high meat diet to test the urine therefor.

The test for indican that I employ is a modification of Jaffé's indican test. Instead of using commercial chloride of lime, I use peroxide of hydrogen. The test-tube which is here depicted is used for the purpose. Chloroform is added to mark 1, which represents 1 cubic centimeter, urine to mark 4, and concentrated hydrochloric acid to mark 7. Shake slightly to cause the acids and urine to mix thoroughly, a moment or two being allowed to elapse before the oxygen is added. Very commonly there is enough oxydase in the urine to start a reaction of indican. In that instance it is unwise to add an oxygen bearing substance. No color of indican being observed, a few drops of peroxide of hydrogen are added, a few moments are allowed to elapse, when if indican is present it will indicate itself by a darkening color. In such an instance the fluid above the chloroform is gently shaken. A moment or two afterwards the entire contents of the tube is shaken vigorously to allow the chloroform to extract the indican from the urine-acid solution, indican being soluble in chloroform. The blue and red varieties are now in the chloroform and the urorosein not being soluble in chloroform, is observed in the fluid above the chloroform as a rose pink. Thus it will be seen that this test answers for both of the indican pigments as well as urorosein.

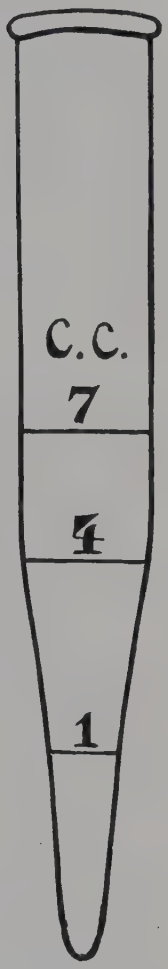


Fig. 45.—Author's test-tube for quantitative estimation of indican and urorosein, which also answers as a fairly accurate quantitative method.

The presence of an increased quantity of indican, or urorosein in the urine requires immediate attention on the part of the clinician. Usually after the bowels are unloaded a decrease will be noted in the amount of indican—sometimes it will disappear. At other times, however, even though the bowels have been made to move thoroughly, no decrease is noted in the quantity of indican. The symptoms in connection with intestinal toxemia in which indican is present are

mentioned in connection with that subject. It need only be mentioned that the test is of much value in clinical work.

In addition to the amount of uric acid in the sediment of the urine is calcium oxalate. While it is true that calcium oxalate crystals in considerable amount are met with in what appears to be normal urine, at the same time it has significance in connection with a metabolic disturbance somewhere in the body. In my experience calcium oxalate, representative of a condition which was recognized long as oxaluria, and which has occupied a debatable ground in medicine, is a clinical condition in which there is breaking down of tissue and in which the connective tissue of the body is interfered with. In gastro-intestinal work the examination of the urine for the total amount of oxalate has considerable significance, and the oxalates can easily be precipitated out of urine by the addition of alcohol.

Cholesterin crystals are occasionally found in urine, and have significance in connection with a condition known as cholesterinuria, particularly in the metabolic condition in which the formation of gall-stones is common. Occasionally it must be examined for in connection with hepatic and biliary disturbances.

Casts are commonly met with in urines of people who have gastro-intestinal disturbances. Their significance depends upon their number and character or any other findings in the urine. It may here be mentioned that hyaline casts, often in increased quantities, are expressions of kidney irritation, and sometimes they are met with in the presence of small amounts of albumin, and one must be careful not to make a diagnosis of nephritis, because commonly when the gastro-intestinal condition is cleared up both the casts and perhaps albumin tend to disappear. Lastly may be mentioned that echinococcus elements are sometimes met with in urine, particularly the free scolices. Of course when they are met with, echinococcus of the urinary tract and kidney is suggested. I have on two occasions met with the embyros of filaria sanguinis in urine, and in another the egg of Distomum haëmatobium. I have also met with one urine containing trichomonas intestinalis from an intestinal infection of that type.

In addition to sulphuric acid, glycuronic acid may be found. The latter may be found alone. Thus are formed, on the one hand, indoxyl-glycuronic acid; skatoxyl-glycuronic acid; cresol-glycuronic acid; phenol-glycuronic acid; and, on the other, corresponding combinations with sulphuric acid, the so-called ethereal sulphates. That a part of these absorbed basic substances is destroyed by oxidization seems probable, another part undergoes different chemical trans-

formations (hippuric acid, indolacetic acid, etc.), but the largest portion is excreted by the kidneys as above, ethereal sulphates and glycuronates.

Except in cases of nephritis, indicanuria affords a fair index to the absorption of indol, which is formed from tryptophan mainly in the lower small intestine, but it bears no direct relation to the absorption of phenol, whose formation from tyrosin in the colon is usually slightly in excess of that of indol. While, as a rule, an excessive indol formation is attended by an excessive production of all the other above mentioned putrefactive products, it will readily be seen that owing to a difference in the character of the protein ingested, and possibly to a difference in the location of an intestinal lesion impeding peristaltic propulsion, the quantitative relation between the indol and the phenol productions may present considerable variations. As evidence of intestinal putrefaction—barring cases of putrefactive abscesses—a urinary test that will indicate the absorbed quantity of these two or more benzene derivatives is, therefore, ordinarily of greater utility than the test which represents but one factor. For such a purpose Folin's test for ethereal sulphates stands unchallenged in scientific value, but its execution demands considerable time, technique, and equipment, which necessarily confines its use to the larger laboratories. The glycuronates, which are capable of intense color reactions, are better suited for simple clinical tests.

The easiest clinical test is that suggested by Askenstedt[6] which combines simplicity with a fair degree of accuracy. The urine to be examined is diluted with water until its specific gravity is reduced to 1.001. For example, if the urine has a sp. gr. of 1.015 (correction being made for temperature), dilute 1 cubic centimeter of urine with 14 cubic centimeters of water; if the sp. gr. is 1.021, dilute 1 cubic centimeter with 20 cubic centimeters of water. (Diabetic urine should be diluted until its urea content is 0.1 per cent.) To 10 cubic centimeters of the diluted urine in a test-tube add one or two drops of a 1 per cent. solution of alpha-naphthol in glycerin (alcohol impairs the test), and then 10 cubic centimeters hydrochloric acid, sp. gr. 1.19. Mix by turning the tube over once or twice. Then let the tube stand in a dry, cool place for about twelve hours, after which the reaction is noted in reflected light. This is best done by holding a white surface, as, for example, a white blotter, behind the tube. Normally the fluid will remain colorless or show a mere suggestion of blue. If glycuronates are present in excess, there will appear a proportionate blue color, tinged with red. The depth of the color may be denoted by the customary use of plus signs, medium blue, the deepest color

observed in the above dilution, being designated with ++++. If greater accuracy of estimation is desired a standard solution of indigo blue, containing some indigo red, in sulphuric acid, may be employed by dropping it into another test tube of the same size, containing 20 cubic centimeters of water. The tube receiving the standard solution is turned over after each drop is added and comparison of the two tubes is made, this procedure being continued until the color is as nearly as possible the same, when the number of drops added is recorded. In twenty-four hours, or later, a slight cloudiness, with fading of color, appears, ultimately forming a sediment.

It must be kept well in mind that the production and excretion of glycuronates are greatly augmented by ingestion of the following drugs, the toxicity of which becomes neutralized by paring with glycuronic acid; chloral hydrate, camphor, turpentine, morphine, menthol, arsenic, acetanilide, antipyrin, benzol, chloroform, curare, hydroquinone, naphthalin, phenol, resorcin, salicylic acid, salol, sulphonal, trional, and thymol. These remedies should, therefore, not be employed before the urine is collected for examination. It seems probable that a comparison of the reaction for glycuronates with the result of a reliable indican test will afford a valuable indication in cases of suspected poisoning with any of the above drugs, but opportunities for a clinical demonstration of the suggestion have not been afforded me.

In estimating the clinical value of the test it must be remembered that the excretion of an unusual amount of glycuronates or indican may be consistent with health. So long as the liver is functionally adequate to neutralize the entire amount of the toxic materials brought to it by the portal vein, little or no harm results, but a constant overstimulation of any function of the body tends to ultimate insufficiency, and a constant excessive production of indican and glycuronates is a positive signal of present or approaching danger.

References.

[1] Crohn: B. B. Crohn, M.D., Eighth Int. Congress of Applied Chemistry, vol. xix, p. 73.

[2] Nuttall and Thierfelder: "Thierisches Leben ohne Bakterien im Verdauungskanal." Zeitschr. f. physiol. Chem., xxi, p. 62, 1896; xxiii, p. 231, 1897.

[3] Levin: "Bakteriologische Darmuntersuchungen," Skandinavisches Archiv. f. Physiol., xvi, p. 249, 1904.

[4] Harden: "The Chemical Action on Glucose of the Lactose-fermenting Organisms of Feces," Journ. of Hyg., v, p. 488, 1905.

[5] Conradi and Kurpjuweit: "Ueber die Bedeutung der bakteriellen Hemmungsstoffe fur die Physiologie und Pathologie des Darms," Münch. med. Wochenschr., lii, pp. 2164, 2228, 1905.

[6] Askenstedt: Jour. of Lab. and Clin. Med., February, 1918.

CHAPTER VI.

Roentgenographic Examination.

No method of examination has done more for abdominal work than the development of the Roentgen method of investigation of the gastro-intestinal canal. To this end the rapid development of apparatus and perfection of technique have contributed much. The use of the vertical screen apparatus for fluoroscopic examination has been of marked assistance in noting details of changes in the anatomy in the gastro-intestinal tract. Any form of energizer answers for the purpose, there being a number of different apparatuses now readily obtainable. What is required is a perfect transformer, a Coolidge tube, a prone and vertical screen apparatus, and an arrangement for the protection of the examiner. The table I use is that devised by Cole in which the fluoroscopic observations are made at right angles, and well within the possibilities of protection. However, the direct forms of fluoroscopic examination in which the tube is below the patient and the screen above answers better in individual examination, such as the movability of the pylorus, the movability of the cecum, adhesions binding down various areas of the gut, etc. Intensifying screens are particularly advantageous because they shorten the time of exposure and in the case of movements such as are constantly going on in the gastro-intestinal canal, the shorter the exposure the better the plate.

In order that comparisons be made on a uniform basis, the X-ray work should be along the lines of a customary routine. This routine may in individual instances be changed where a special examination is required, but as a rule it answers best for general work. In the course of time the majority of persons doing a considerable amount of abdominal X-ray work acquire a routine which satisfies them.

At the beginning of an X-ray examination the stomach should be empty. This may be accomplished by means of tubing and lavage or long enough after a meal. It is my custom to have the patient take a purge the day before the examination and then arrive at the X-ray department four or five hours after his breakfast, when the first observations are made; then to return in six hours when the ileocecal region is examined; then in twenty-four hours from the first

appearance when X-ray plates are made of the colon without a clysma, these followed by a clysma, the observations being made from fifteen minutes to a full hour following it. Plates up to 3 or 4 days following the barium by mouth are required in some instances.

The best form of opaque salt to use is that of chemically pure barium sulphate, about 90 grams or 3 ounces being used as an instillation in the stomach and the same amount being sufficient for colon clysma work. The vehicle for this had best be some form of fermented milk for the stomach ingesture or a cereal or potato starch paste of some sort. That which I use for a clysma is a heavy solution of syrup of Acacia and water, the entire quantity with the barium made up to about 20 ounces.

I believe that there are distinct advantages in the examination of the digestive tract by means of a screen. By its use exact information may be obtained as to mobility and flexibility of organs, the phenomena of peristalsis and anti-peristalsis, the nature and permanence of irregularity in contour, and the effect of palpation and respiratory movements in the various positions. The screen also affords an excellent opportunity for inspection of the chest and abdomen to note the numerous lesions which might affect the digestive tract reflexly.

The fluoroscopic examination must be conducted in a darkened room, and it is best for the examiner to have remained in that room for some time before beginning the investigation. If occasionally one has to go out into a lighter room the use of Uphose glasses of the deepest green are to be recommended. In the fluoroscopic examination the male patients are stripped to the hips, while women may be allowed to wear a kimona of thin material providing it is devoid of buttons or ornaments. The underskirt should be dropped below the trochanters. The operator should endeavor to secure the greatest amount of protection consistent with efficiency. He should wear gloves of leaded rubber or other opaque material and should protect the genitalia with an apron of leaded fabric. As to the patient he is saved by the protective features of the apparatus and the brevity of the examination. I am also a believer in the Cole form of serial radiography, although of late the use of the screen has come so prominently forward that it has largely taken the place of the plate method. For most of the examinations I employ an anode distance from the X-ray plate of 60 centimeters. Occasionally, however, in gall-bladder work the distance is increased to about 75. The plates are the most rapid procurable, and the developer is the usual form of metol-hydrochinone, the fixing bath being "acid-hypo."

The main part of radiographic work is the interpretation of the X-ray plates. Here one has to deal with shadows, shadowed effects and signs of normal, and pathological conditions, and naturally these signs vary in their degree, some being more or less direct showing as definite and permanent additions to or subtractions from the normal contour. Others are rather indirect, revealing only perversion of function, and the seat and nature of the pathologic change can only be inferred or must be searched for elsewhere. The danger of making a diagnosis based on only a few X-ray plates deserves particular condemnation, because a single plate shows only a single phase and a single angle of view. Of course the reason for this is a saving of expense, but if one is going to engage in X-ray work, obtaining therefrom all that is possible for the X-ray examination to give, it is necessary that the examination be made complete and thorough.

There is a large amount of debate still going on as to who should interpret the X-ray plates to the best interest of the patient. Having engaged in X-ray work almost from its beginning, all my work is done in my own X-ray department and I do the interpreting after having attended to the matter of the history, the performing of the physical examination and all that pertains to the diagnosis of the case including that of the laboratory procedure. Time and time again I have seen patients with X-ray diagnosis which were not substantiated, either in a clinical way or at the operation. Therefore I think that unless a clinician has had extensive X-ray experience and training, it is better for him not to interpret the findings himself, but take those from a Roetgenologist who has had somewhat of clinical experience, either in connection with various hospitals, or clinics, etc., or because he has had such experience before he engaged in Roentgenographical work. Even then, the clinician and the Roentgenologist should make a joint examination and go over the respective findings at a personal conference.

Since the X-ray work of the esophagus and stomach is included in my textbook on those organs, the point of procedure will be to begin with the small intestine.

In most instances the small intestine can be studied as a supplement to and during the examination of the stomach. This pertains to matters such as hypermotility, mostly gastro-enteric in type; the matter of the transit through from the stomach to the ileocecal valve; matters pertaining to ileal stasis—all are possible of being noticed following the stomach examination, that of the colon being made the next day and the days following.

One can easily see the passage of the opaque meal through the pylorus, it going about the horseshoe of the duodenum and disappearing behind the stomach, appearing again in the first part of the jejunum, and advancing down into the corners of the ileum. The beginning of the examination may be performed with the patient in a standing position but that which pertains to the lower coils of the ileum had best be done in the prone.

Irregularities of the duodenal contour have as the most common causes spasms or scar contractions from duodenal ulcer and pericholecystitic adhesions. One must be careful to remember that the inner contour of the first part of the duodenum is sometimes indented from pressure against the spine. Evidences of obstruction are generally not hard to observe. These include localized and permanent narrowing of the lumen with dilatation proximal to it, and delayed motility.

DUODENAL ULCER.

If the examiner is experienced, the screen examination alone will be very decisive in noting the presence or absence of duodenal ulcer. My experience, after having made a few mistakes through depending upon the screen examination alone is that the serial radiography method is best in making this diagnosis.

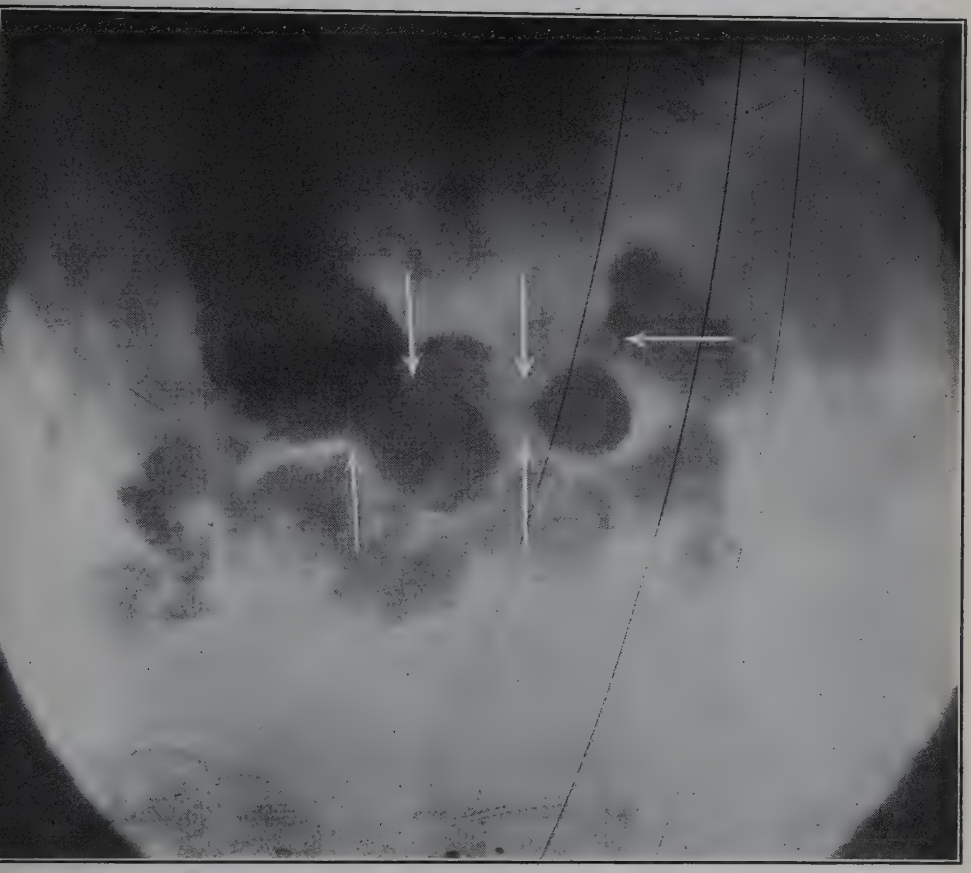
The Roentgenographic diagnosis of duodenal ulcer depends upon the following:

1. Deformity of the duodenal contour, more especially the first portion. One must be careful here not to mistake a deformity of the outline of the duodenum in certain cases of spasm and those irregularities which are due to the tugging of adhesions that are fastened onto the duodenum. Occasionally one can see the niche type in which a barium-filled recess projects from the bulbar chamber. This however is distinctly less common than are instances of perforating ulcer of the stomach. A diverticulum may be present in the duodenum as well as elsewhere in the gastro-intestinal tract, in which instance there would be a sac-like addition to the bulb proximal to its side. Quite often in diverticulum of the duodenum an ulcer is associated with it. As mentioned above, the spine may deform the bulbar outline, especially at the inner border and by using both the screen and plate methods the cause would be evident.

2. The alteration in gastric tone, the most common change being a hypertonus. According to Carman[1], this together with hyperperistalsis and hypermotility, constitutes a triad of "hypers" which was early recognized as being rather strongly indicative of duodenal ulcer.

PLATE III

X-ray plate of duodenal ulcer. Upper arrow points at site of lesion; lower, an incisure spasm opposite it. Confirmed by operation. (X-ray by author.)



X-ray plate of case of duodenal ulcer, showing marked gastrospasm. The vertical arrows pointing to stomach spasm, the horizontal one to site of lesion in duodenum. Confirmed by operation. (X-ray by author.)

PLATE V

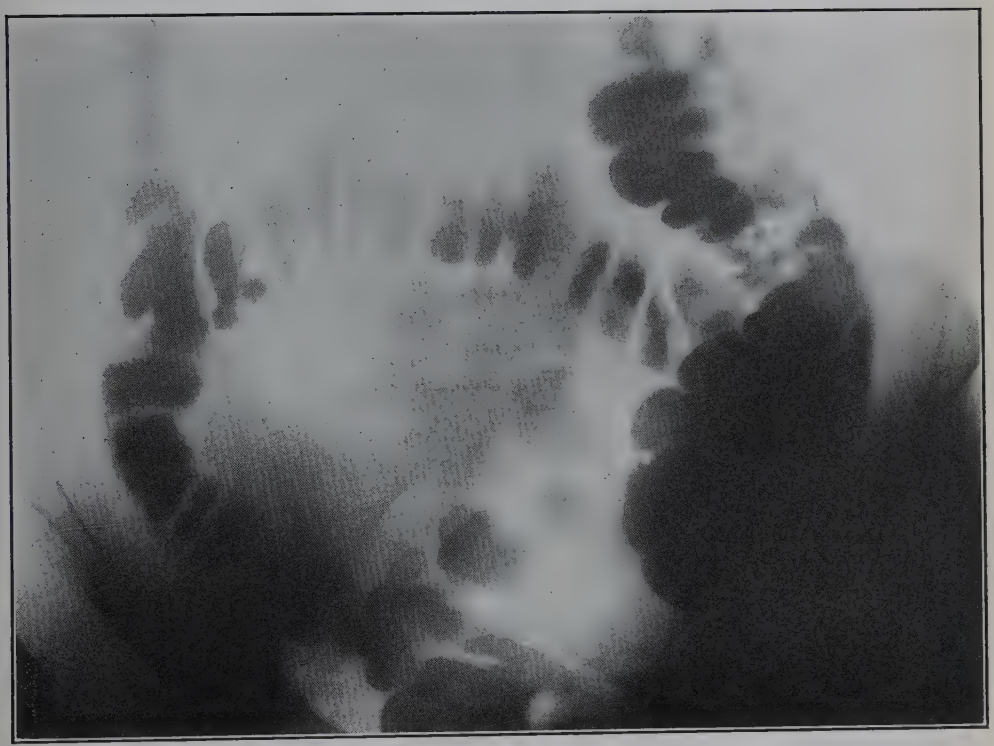
Arrow points to residual barium in ampulla of Vater in case of cholelithiasis, proven by operation. Also 6-hour residue in stomach. (X-ray by author.)

Normal colon. (*McMahon* and *Carman.*)

PLATE VII

Prolapse of the colon, and of both the hepatic and splenic flexures also. Small normal appendix also showing. (X-ray by author.)

PLATE VIII



Mobile cecum, freely displaceable by hand under the screen. (X-ray by author.)

Hyperperistalsis is notable in perhaps 60 per cent. or more of duodenal ulcers. It varies from a slight increase to a tempestuous energy of contraction. It is most exaggerated in the obstructive cases but it also occurs in non-obstructive ones—in fact those instances of the most marked activity I have seen have been in the non-obstructive cases. It must be remembered in this connection that peristaltic waves are not commonly seen in the first part of the duodenum—in fact, nowhere from the pylorus downward until one reaches the colon. Hypermotility is given as an important point in connection with the diagnosis of duodenal ulcer but in my opinion is not so much to be depended upon as is the direct method of examination, namely, that of change in the contour.

3. Duodenal ulcer is frequently associated with spasmodic manifestations in the stomach. These manifestations may be so marked and hold so persistently that one may easily make the mistake of supposing that the ulcer is situated in the stomach instead of in the duodenum.

4. Tenderness located at the duodenum is included as one of the manifestations of ulcer signs. In my opinion it may be present and be of some diagnostic value, but in most instances it is not, and its absence should not rule against the presence of an ulcer.

HYPERPERISTALSIS OF THE SMALL INTESTINE.

Not uncommonly cases are seen in which the emptying of the small intestine is distinctly faster than should be. I have seen instances where the head of the barium column is in the mid-transverse colon in an hour and twenty minutes. Of course in such instances there is usually a hyperperistalsis of the stomach also, but I doubt if the hyperperistalsis of the stomach could account for the rapid transit through the small intestine. This is a clinical condition of some significance and should be mentioned in connection with Roentgenographic work.

Occasionally, too, can be observed a hesitancy of the barium mass somewhere in the small intestine thus denoting the presence of a stricture. Lane's kinks, S shaped routes of the small intestine in close proximity to the ileocecal valve are generally easily diagnosed.

Another interesting Roentgenographic finding is the residue of a small bit of bismuth in the region of the ampulla of Vater. I have seen at least a dozen of such instances, and while in the majority of them it is not possible to say whether there is a diverticulum which may have occurred at that site or whether it is due to a large ampulla

to which some of the barium had gained access and therefore perhaps having significance in connection with gall-stone diagnosis, as no doubt the condition exists, it should be mentioned in this connection.

THE LARGE INTESTINE.

Examination of the colon either by the ingested meal or by the clysma may be employed. My technique is to employ both—the barium by mouth to demonstrate the advance in connection with the diagnosis of colonic stasis; the barium by clysma to give one more perfect pictures of the anatomy of the colon. In the instance of a clysma it is desirable to make a screen observation while the clysma is running in—perhaps also to engage in more or less manipulation during the investigation.

To describe a normal colon is quite impossible. There are so many dissimilarities in different individuals that the allowance for exceptions has almost forbidden a description of what may be termed normal.

A point important to recognize is the condition of the colon in the standing and prone postures, disparity between the height in the splenic and the hepatic flexures, loops and redundancies; sagging of the transverse colon, matters pertaining to dilatation—as to whether the dilatation is only on the right side or involves that of the entire colon; the regularity of the haustral contractions and the depth and number of them—as to whether also they are found present in the ascending colon and whether they are present in the descending colon and sigmoid. Palpatory manipulation pertaining to points of fixation are important. The hepatic flexure as a rule is susceptible of slight displacement, although the splenic flexure is generally fixed.

Among the abnormal points in this connection may first be mentioned those due to changes of position in the colon. Arrested developmental migration may result in a high placed cecum, or the entire colon may still lie to the left of the spinal column, the so-called non-rotation. By failure of the cecum and ascending colon to rotate on their long axes the ileocolic juncture may be found on the outer aspect of the cecum instead of the inner.

Displacement of any part of the colon may be produced by extrinsic tumors of every sort including those of the liver, pancreas, spleen, etc., by adhesion bands, by pregnancy, by psoas abscess. It would be important to observe the relative height of the two flexures, because commonly the hepatic flexure is found displaced downward in cases of prolapse. Also it is desirable to note the

position of the transverse colon, which often is festooned and the large part lies perfectly within the pelvis.

Another point is the irregularities of contour, namely the filling defects, which may be produced by intrinsic tumors, diverticulitis, or bands of adhesions. One must be careful here not to be misled by filling defects caused by fecal matter, fecaliths, gas, localized spasm, extrinsic tumors, insufficient quantity of enema, etc. There may be cases of actual kinking, or of loops at the angles, observed most commonly in the splenic flexure and in the sigmoid region.

The mobility of the colon is also important, particularly the cecal region in connection with what is known as a mobile-cecum, as described by Wilms. Diminished mobility or even complete fixation is a common sequence of adhesions producing pericolic inflammation. Not uncommonly the cecum is fixed by adhesions from the appendix or from some form of pelvic condition or malignancy. Here comes in the important matter pertaining to the so-called Jackson's membrane, which while not always possible of being diagnosed by X-ray, nevertheless can be occasionally. The only way adhesions can be diagnosed is by the anchoring of the gut at some definite point or the inability to draw two coils of the gut away from each other. As a rule, however, the diagnosis of adhesions of the large intestine is not easy.

One sees various changes in peristalsis of different sorts in cases of chronic colitis; in cases of spastic constipation or neurological phenomena of the abdomen in which irregular haustral contractions may be noted in the transverse and descending colon. Lane has pointed out that the absence of peristaltic movement in the sigmoid and the lower end of the colon is quite characteristic of a toxic state, and that what is known as a straight sigmoid is commonly due to an "auto-intoxication."

Increased length of colon and redundancy, most marked in the sigmoid and transverse portions, is a relatively common finding in constipated persons, and must not be taken as of too great significance because they may be found in perfectly normal individuals. Redundancy of the sigmoid with dilatation has been observed occasionally. Hirschsprung's disease, or congenital idiopathic dilatation of the colon, should be mentioned, in which there is immense dilatation throughout, and this is strikingly shown by the X-ray. Dilatation of the cecum, ascending and transverse portions, with evident loss of tone, is not infrequently noted. Dilatation of any part of the colon, proximal to a stenosis, may be evident. Spastic narrowing of the colon, particularly in the transverse, descending and sigmoid portions

is noted occasionally in cases of constipation, but it may also be observed in a colitis, even with frequent stools. A narrow unhaustrated colon is often seen in cases of granular or ulcerative colitis.

Hypermotility of the colon is seen typically in association with non-obstructing gastric carcinoma, in which condition the head of the meal may be in the descending colon or even in the rectal ampulla in six hours. It is also observed in duodenal ulcer, achylia and diarrheic conditions. Hypomotility with decidedly slow progress of the meal through the colon, may be due either to organic obstruction, or as is most common, to functional impairment of peristalsis commonly in association with an intestinal toxemia. This in the small intestine is designated as ileal stasis; in the large intestine as colonic stasis.

The most important sign of cancer of the colon is a filling defect, a local irregularity of contour produced by the jutting of the growth into the intestinal lumen, also perhaps by contracture of the infiltrated wall, and probably to some extent by spasm aroused by the neoplasm. Important, too, is the manifestation of obstruction, or blocking at a definite point. One must remember that the most common site of carcinoma of the colon is at the flexures and in the rectum.

Except in the presence of a peridiverticular abscess when the diagnosis can be assumed, there is no other way of diagnosing diverticulitis excepting by the X-ray. It will then be noticed that diverticula vary in size from that of a pea to a hen's egg. They are usually round or ovoid and most often sessile, though occasionally pedunculated, and most commonly found in males, particularly those inclined to obesity. They have no great diagnostic importance, although their presence when noted should be made known to the patient so that should an acute abdominal attack occur at any time the knowledge by the patient of the presence of diverticula might be most valuable.

Secondary to pulmonary tuberculosis is tuberculosis of the ileocecal valve region, wherein the condition might be that of a soft ulcerative process or hyperplastic type of pathology, and sometimes a combination of the two. Filling defects in the region of the ileocecal valve, perhaps with a palpable mass, constitute the chief manifestations. Naturally the first impression would be that of cancer, but in the presence of defects in this region one should hesitate and make sure that no tuberculosis of the lungs or elsewhere exists. Other than these filling defects I do not believe that there is any definite way for diagnosing tuberculosis of the colon. Some time ago it was suggested that one of the characteristic findings was an emptiness of the ascending colon. This, however, in my experience has been

PLATE IX

Chronic appendicitis. Arrows show the appendix kinked at the arrows, and the tip is adherent under the cecum. Confirmed by operation. The absence of peristalsis and straight line sigmoid suggests an intestinal toxemia. (X-ray by author.)

proven fallacious in enough instances to throw it out of consideration altogether. The most important point is the filling defect or some change in the contour of the gut.

The Roentgenographic study of the appendix offers a field of work that is worth while. Of course the acute cases never come under observation in the X-ray room, but the subacute and chronic varieties commonly do. An appendix which remains visible for more than a day or two following the bismuth examination in my opinion, with that of Case, is commonly an appendix which does not drain properly and is dangerous. Practically all of them should be removed. It is not possible to demonstrate the appearance of the appendix in every individual, but in the larger proportion of them it is. One should hesitate, however, in diagnosing chronic appendicitis on the appearance of irregularities in the bulbar end, possibilities of adhesions to the ileum and so forth, because commonly an appendix like a worm, is capable of having most extraordinary movements and gives on X-ray plates shadows which may be deducted as pathological. As a rule, however, if the X-ray work is carefully done the following is to be taken into consideration in connection with the diagnosis of appendicular conditions; first, concretion in the appendix; second, kinking; third, malposition; fourth, adhesions about the appendix and cecum; fifth, retention of barium in the appendix; sixth, pressure-tender points related to the appendix, particularly at the base.

Polyposis of the colon, a very rare condition, is almost impossible to diagnose with any degree of accuracy by the X-ray.

Insufficiency of the ileocecal valve is not an uncommon finding in the X-ray examination, particularly by the clysma method. Much attention has been drawn to this condition and while it possesses a practical significance, most times it is of no importance. It is not uncommonly found in perfectly normal individuals and can be produced physiologically under conditions of starving, etc.

The diagnosis of foreign bodies within the intestines is a matter of some importance and needs only to be mentioned. They are easily located providing the foreign bodies contain a substance which is more obstructing to the X-ray than the tissues. Most often, fortunately, they are metal articles and thus can be easily observed, in which instance no opaque mixture would be used but just a general search of the abdomen followed.

It is occasionally important to inject external fistulæ to see the length of them, and also the exact part of the bowel from which the fistulæ originate, as well as the extent of their ramification. This is done by injecting a bismuth paste to visualize the fistula, and

taking the plates promptly afterward. In my experience the effort to fill fistulæ from within the gut is rather difficult. Not uncommonly one meets with a case of peridiverticular abscess which has had a fecal fistula for a number of years, in which there are present, attacks of cramps and pain most distressing to the patient. In those instances giving the bismuth by mouth and expecting that it will probably find its way into the fistula tract is a very uncertain procedure, yet it is the only one at our command. In eight of such cases I managed to fill the fistula's tract from the inside 3 times. Injecting bismuth from the outside is preferable.

GALL-STONES, AND DISEASE OF THE GALL-BLADDER AND LIVER.

The degree of decision with which gall-stones can be diagnosticated by the Roentgen-ray is still in debate. Roentgenologists report all the way from 20 to 80 per cent. of positive findings. In my own experience the definite gall-stone cases in which the plates showed definite presence of stones represented 42 per cent. in over four hundred cases. Caldwell[2] expressed the truth when he said: "If we only make enough plates and make them well enough, we can obtain suspicious shadows in the gall-bladder region of any normal individual. The interpretation of gall-stone plates, therefore, becomes a matter involving not only skill and judgment, but temperament as well. In no other field of Roentgen diagnosis is the personal equation of the observer more important. This, I believe, is the best explanation of why some observers report as high as 85 per cent. and others as low as 5 per cent. of successes in these examinations. The greatest danger of the X-ray is in faulty interpretation. These faults usually consist of reading into the plates something which is not there. The clinical indications of gall-stones are fairly accurate, and it is likely that stones are present in at least 50 or 60 per cent. of those cases submitted to X-ray examination. Of these perhaps as many as one-tenth give perfectly definite, reliable X-ray shadows of gall-stones. In the other nine-tenths of the cases examined, some of the X-ray plates will show hazy and suspicious shadows, many of which are due to gall-stones. If we consider all these hazy and suspicious shadows corroborative evidence of gall-stones, the operation will vindicate us in the majority of the cases, and we may easily overestimate the help actually obtained from the X-ray plates. We must be very careful lest the clinical knowledge we have of the patient may lead us to accept as evidence of gall-stones, X-ray shadows such as might

be found in some of the X-ray plates of any normal individual." He therefore warns against regarding as proof of gall-stones "any X-ray shadow that does not present characteristics distinguishing it definitely from other shadows, and which has not the sharpness of outline commonly found in other calculi."

The important points in the possibility of demonstrating gall-stones include composition, size, number and situation, their developing media—that is as to whether they have been there for a long time and have a calcium veneer; the amount of obstruction by liver tissue; the thickness of the patient; the thoroughness of his preparation, and the Roentgenographic technique. Stones of large size are usually not difficult to demonstrate on a plate, but so many cases of gall-stone disease have stones that are minute and few in number that they perhaps are not possible of being diagnosticated. It should be mentioned that pure cholesterin stones have but slight density and are most difficult to show. The mixed type of stones, those containing cholesterin, bilirubin and calcium salts, are more common, and their density is proportionate to the amount of contained lime. In other words, one gets the shadow from the lime contained in the stone and not of the stone itself. This makes it possible for stones which have been in the gall-bladder for a long time and have a large deposition of lime salt to cast a deep shadow. Such stones, too, are generally large ones and unfortunately they comprise only, according to Carman, 1 per cent. of the total.

In my opinion the diagnosing of gall-stones by means of the X-ray is the most difficult part of X-ray work. A preliminary clearing out of the intestinal canal is important, and it is best to make the examination before the giving of an opaque meal, although at times one may see gall-stones with an opaque meal in the stomach when the proximity of the gall-bladder to the pyloric end of the stomach is kept in mind. The most important point of the technique is to make a number of X-ray plates—of course fluoroscopic examination for their detection is out of the question. Not uncommonly in a dozen or more plates no evidence of gall-stones is present, whereas in one or two following they are as manifest as anything can be. Therefore the most important point of the technique is to make a large number of plates and at different angles, using a rather small cone. The plates should be made during suspended respiration to avoid blurring by movement, because the merest movement throws out the possibility of making a diagnosis. Thus it is important that an intensifying screen be used, because the exposure should be made as short as possible. It is essential also that the tube be a moderate

vacuum, inclined to softness rather than hardness and over-exposure should be avoided. In my experience about a four inch vacuum is best. The patient should be prone with the abdomen downward.

Shadows of the gall-bladder must be differentiated from those of renal calculi. The latter are usually branched or irregular and of greater density than the gall-stones because they contain phosphorus, thus casting a more intense shadow. A point of differentiation is a good detail of the outline of the kidney, or possibly the outline of the gall-bladder. Calcified lymph-nodes may be mistaken for gall-stones but the former seldom show in clusters and are often irregularly shaped. Deposits of lime-salts in the ribs generally show irregular streakings and are directly in line with the rib shadow. Fecaliths and fecal matter in the intestines may produce shadows somewhat resembling those of gall-stones, and for this reason the bowels should be thoroughly evacuated beforehand. Finally the admonition is given that unless one can be positively sure that gall-stones exist, it is best not to allow imagination to carry one too far into a definite diagnosis by X-ray.

CHOLECYSTITIS.

Cases of acute cholecystitis rarely come under X-ray observation, but it is not uncommon to arrive at a diagnosis of chronic cholecystitis by X-ray means. The gall-bladder when thickened, or when it contains inspissated bile, occasionally casts a shadow if the patient is not too thick and other circumstances are favorable. As pointed out by me, and later more distinctly brought out by George, when a gall-bladder is manifest upon an X-ray plate, it can be judged as sufficiently pathological for operation. This in my opinion is the most valuable sign in this connection, to which may be added as an indirect sign, gastro-spasms; six hour residue in the stomach or duodenum; the fixation of the pyloric end of the stomach, duodenum or hepatic flexure; the drawing of the stomach to the right by adhesions; or displacement to the left by a mass about the gall-bladder; hyperperistalsis in the stomach; the demonstration of a Riedel lobe, which commonly has a diseased gall-bladder associated with it; localization of a tender point over the gall-bladder region and the absence of any demonstrable lesions in the stomach and duodenum, etc., to account for symptoms which suggest some form of chronic trouble.

A six-hour residue in the stomach or duodenum may occur as a result of pericholecystic adhesion-bands constricting the pylorus or duodenum from the gall-bladder. Occasionally, too, one may meet

with a degree of stagnation in the stomach in cases of cholecystitis without the presence of adhesions. The duodenum may be deformed or stenosed by adhesions, and the contour of the stomach may be roughened. Adhesions may fix the hepatic flexure of the colon, which in my experience is most rare, or the pylorus or duodenum may be so fixed that they cannot be separated from the liver by manipulation. I agree with Case[3], that the presence of a Riedel lobe of the liver, which may be demonstrable after inflation of the colon, is a valuable sign of cholecystitis.

Important in this connection is the point which Carman[4] expressed—namely, "the most common lesions giving rise to symptoms which are often confusingly similar are gastric ulcer, duodenal ulcer, cholecystitis and appendicitis. When the Roentgen findings negate the presence of a peptic ulcer, the field of reasonable consideration is narrowed to the gall-bladder and appendix."

THE LIVER.

Useful information can commonly be obtained by the Roentgenographic method as to the upper border of the hepatic shadow, coinciding with the diaphragm, and the presence of growth or collection of fluid and gas (subphrenic abscess). With a uniform technique constantly employed one not uncommonly can get an idea of the density of the liver from the shadow that it gives, and in pathological livers demonstrate the free border.

An increase of the liver-volume, with general and symmetrical enlargement of its shadow may be due either to hyperemia, or to an actual hypertrophy resulting from various causes, including amyloid degeneration, cancer, syphilis and cirrhosis. The small liver of atrophic cirrhosis is distinguishable with difficulty. The liver may be ptosed, in which case separation of the upper hepatic border from the diaphragm may be apparent. The normal rise and fall of the liver with expiration and inspiration is sometimes evidently diminished or absent when fixation has resulted from inflammatory processes, either in the chest or upper abdomen. Whenever the lower lobe of the liver cannot be demonstrated the presence of gas in the hepatic colon serves to give an idea of its location, the liver occupying the area between the height of the gas and the edge of the diaphragm.

By the introduction of oxygen gas into the free peritoneal cavity both liver edges are plainly discernible. The same may be said of the entire spleen, the body of the uterus and in some instances the ovaries. Even small quantities of fluid are strikingly shown by this method. It is accomplished by the introduction of warmed oxygen by

means of a canula, the plunge made in the midline halfway between the umbilicus and the upper level of the pubic bones. One must be careful that the urinary bladder is empty. The gas is run in slowly until a considerable distention of the abdomen is accomplished. Fifteen minutes time is allowed, after which the X-ray plates are made. While not original with them, to Dr. Stein and Dr. Stewart of New York City should be given the credit of the perfection and the introduction practically of this addition to X-ray examination of the abdomen.

References.

[1] Carman, R. D.: "The Roentgen Diagnosis of Diseases of the Alimentary Canal," p. 403.

[2] Caldwell, E. W.: "The Safe Interpretation of Roentgenograms of the Gall-bladder Region." Am. Jour. Roentgenol., 1915, ii, 816-819.

[3] Case, J. T.: "Roentgenoscopy of the Liver and Biliary Passages, with Special Reference to Gall-stones." Jour. Am. Med. Assn., 1913, lxi, 920-24.

[4] Carman, R. D.: "Diseases of the Gall-bladder and Liver."

CHAPTER VII.

Chronic Excessive Intestinal Toxemia.

PUTREFACTION, INDICANURIA, AUTO-INTOXICATION.

INTESTINAL FOOD-BACTERIA TOXIC STATES.

The subject of chronic excessive intestinal putrefaction, as well as the mixed and saccharo-butyric forms, is bound up in such a number of factors that an analysis of the condition as a clinical entity is not easy. In considering the subject we have to deal with the age of the individual, the infection of foods, combinations of foods, the effects of foods upon the bacteriology of the intestinal canal, the bacteriology of the intestinal canal independent of whatever foods are taken, the economic factors pertaining to life, the sociologic conditions under which the individual lives, the status of secretion in the stomach, those poured into the small intestine, those of the intestinal mucous membrane, the status of motility in the stomach, small and large intestine, the rate of transit through the intestinal canal, oral and faucial sepsis in relation to a direct infection of the gastro-intestinal canal, various infective conditions such as those locked up in the gall-bladder and appendix, as they may infect the intestinal canal, matters of bands, kinks, adhesions, etc. It will thus be seen that what might be classed as intestinal toxemia is far from a simple condition, even in the individual case.

It serves no purpose to say that because certain toxic bodies are not recoverable from the urine or elsewhere that no such condition as intestinal toxemia exists. When one keeps in mind the many causative factors mentioned and the various processes that have to do with resorption and assimilation, the oxidizing processes in the general economy, matters pertaining to elimination, the deficiencies that exist in biologic knowledge, the deficiencies in analytical physiological chemistry, the fact that no definite substance is recoverable is no proof against the existence of these conditions. All around us there are instances of people who for many years have conditions that can only be due to disorders of this kind. These individuals are immutable facts and while scientific medicine has not advanced to the extent of making possible the recovery of any definite substance and by so doing arrive at a definite diagnosis, the individuals still are there, and with the even suggestive diagnosis, the successful handling shows

definitely that these conditions are distinct disorders. One might as well say that because he was not able to handle a portion of the substance of the sun no such thing as the sun existed, even though the entire world depends upon it for life. There are indirect methods of knowing that this luminary is part of the solar system, and so there are indirect methods also of knowing that a condition such as intestinal toxemia exists.

For a number of years this subject has been the most debatable ground in medicine, and those who have contended the most against the existence of such disorders are those who know the least about them either by clinical or laboratory experience. There are many persons in medicine who because they do not understand a certain subject, directly make the statement that no such subject exists. Clinical facts, however, are immutable facts, and if the subject is approached with an open mind and largely from the clinical standpoint, one cannot but arrive at the conclusion that these states are a *bona fide* disorder of mankind and most common.

In 1887 Bouchard[1] advanced his theories on "auto-intoxication" of the intestinal canal. Much, however, that he wrote pertaining to food poisoning, internal strangulation and infection, such as typhoid and cholera, are not worthy of consideration today.

Metchnikoff[2] is considered the real founder of what we might call the "school" of intestinal toxemia. His idea that the colon was simply a breeding place for pathogenic bacteria which caused most of the ills of mankind, and which could be successfully combated by the Bulgarian bacillus, is now an exploded theory. In this connection it may be stated that Metchnikoff is not entitled to the credit of drawing attention to bacteria as having to do with the infection of the intestinal canal. Certainly before 1907, Christian Herter[3] had been working for several years on the subject, and it may be stated that comparing the works of Metchnikoff on the one hand and the works of Herter on the other, those of Herter have stood the test of time, and in my opinion, are the best expositions of bacterial infections of the intestines of man that have yet been advanced.

Following this was the work and the propaganda carried out by Sir Arbuthnot Lane[4], in which he undertook to draw attention to stasis, ptosis and toxemia as having to do with these conditions and strongly suggested operative interference for their alleviation and cure. According to him the primary factors are mechanical. The process begins with an overloading of the large bowel, especially in the right side, due largely to the erect position, and exaggerated by corsets and defecation in an upright sitting position. Stagnation in

the cecum with distention and descent into the pelvis follow; adhesions form across the lower end of the ileum and the appendix in an attempt to hold the cecum in position. The strain on the right colon makes it difficult for fecal matter to pass, and adhesions form to hold the bowel in place. These tend to contract, pulling the hepatic flexure up higher than normal. The same process finally takes place on the left in which the transverse colon sags, drags on the stomach and pulls it down, and adhesions form about the pylorus, duodenum and gall-bladder. The sigmoid becomes distended, shortened and straightened, or much distended in the middle and caught at both ends by adhesions. The adhesions across the lower end of the ileum anchor the ileum forming the famous Lane's kink, the whole process of adhesion formation having, according to Lane, its origin in strain and the resulting "crystallization of lines of strain." Following out his theory, stagnation in the bowel causes abnormal putrefaction of the contents, and the absorption of toxic material produces a degenerative change in all the structures of the body. With this idea he swept in all the ills of mankind throughout the world as directly or indirectly due to stasis, the following conditions being caused (taken from the writings of Lane, Watson,[5] and Charles H. Mayo[6]): Appendicitis, arteriosclerosis, arthritis, carcinoma of the ovaries, cholecystitis, circulatory disturbance, colitis, constipation, curvature of the spine, cystic degeneration of the ovaries and breasts, diarrhea, endarteritis, epilepsy, foul perspiration, floating kidney, gall-stones, gastric and duodenal ulcers, gingivitis, gout, interference with respiration, intestinal obstruction, loss of sexual desire, loss of control of temper, loss of vitality, mastitis, migraine, muscular atrophy, neuralgia, neurasthenia, pancreatitis, pharyngitis, pigmentation of the skin, pyorrhea, renal disease, tuberculosis, uterine versions and flexions, visceral ptosis, and volvulus.

While it is true that all the above conditions can be, and often are caused by intestinal toxemia, the truth is that they are nowhere near such definitely resulting conditions as Lane would have us believe. More than that, the theory of Lane will not stand close analysis, neither from the clinical standpoint nor from the standpoint of results after operation upon people having conditions such as Lane suggested could be relieved or cured by various operative procedures. The trouble with Lane's contention is that he considers the subject entirely from a mechanical standpoint, and the subject is biologic. He dealt with a biologic subject in which he advanced drainage-surgery for its correction, with the idea that by changing the fecal current he could alleviate most of the disorders of mankind after God

had given mankind the intestinal canal that he did. Still, it is true that stasis and ptosis are factors in connection with toxemia, although the method which Lane suggested for their cure is not the best. I feel that after all Lane's propaganda there is perhaps 10 per cent. of worth in what he has done. The rest of it immediately began to fall when ambitious surgeons throughout the world took his propaganda according as he advanced it and accomplished baneful results from the operative procedures he suggested. Then in 1913 Kellogg[7] published his papers attempting to demonstrate that practically all of Lane's list of diseases with a few more added, were due to the incompetency of the ileocecal valve, allowing reflux of the contents of the colon into the ileum, suggesting at the same time as a method of treatment the surgical procedure of tightening the valve a bit to prevent the reflux, and advancing the opinion that this was a safer method of procedure. Kellogg went so far as to say that a Lane's kink was due to incompetency of the ileocecal valve, and this in face of the fact that many perfectly normal individuals have Lane's kinks and incompetent ileocecal valves without any symptoms from either one, or even from a combination of both.

In 1913 Martin,[8] of Philadelphia, and I showed that Lane and Kellogg were wrong, and that the only primary cause of trouble for which surgery was warranted is a stenosis, or stricture, somewhere in the enteric canal, although Martin suggested that this stricture was of the ileocecal valve, causing retention of the waste products of digestion in the ileum, and that in certain cases the valve opening should be enlarged by a simple surgical procedure which he describes.

Then came along Case with a paper he read in Chicago showing that many of the abnormalities which could be noted by the X-ray to exist in the right abdomen were really due to obstructive conditions in the sigmoid.

Up to the present then we have the theory of Bouchard, that of Metchnikoff, and Herter; that of Lane, that of Kellogg, the one of Martin, the one of Case, to which may be added other theories, such as the one of Keith[9] that intestinal stasis is always secondary to a neuromuscular disorder of the bowel, due to a disturbance at the nodal points and this is the theory that I partly believe in, with the addition that when definite obstruction exists, this may be a cause also. Later came the theory of Beveridge who thought that these disorders were due to disturbances in the internal secretion of the ductless glands, and that these could produce disorders such as putrefaction in the intestines. And lastly the one of Goldthwait in which he ascribed anatomic and mechanistic causes for practically the same list of dis-

eases that Lane had given with a few additions, such as goiter, eye trouble, mental disease, diabetes and race degeneration, and stated that they were due to anatomic anomalies in the skeleton and improper posture resulting in pressure on various organs, causing a multitude of troubles, many of which might be relieved or cured by a change in posture. He included in one of his statements "One occasionally sees convulsions stopped instantly by a mere change of position," to which I would add that in epilepsy, frequently a change in methods of treatment, and even just the attention of a new physician, will cause the epileptic to be free from attacks for some time. Goldthwait also states in speaking of diabetes "One has seen cases in which after most careful dieting the sugar has persisted, and then after supporting the organs properly the sugar has wholly disappeared." I think, however, that this should not be taken too literally as a cure or a method of relief in any cases of diabetes.

In 1899 Adami wrote instructively on "Latent Infection" and "Subinfection," and again in 1914 an exhaustive article on "Chronic Intestinal Stasis," taking the view that low-grade infections are the primary causes in most of the same much-discussed group of diseases that have been mentioned. Since 1911, Billings, Rosenow, and many others have written on focal infection as the cause of arthritis, appendicitis, gastric and duodenal ulcers, and many other diseases in the above mentioned group.

We have, then, a more or less well-defined group of some forty or fifty diseases and morbid conditions, of difficult to prove direct etiology, and about a dozen men of experience and standing in the profession, each advancing some one condition as the primary cause of all the diseases, and each presenting series of cases and clinical evidence to prove his contention. Manifestly they cannot all be right, and yet, they are not all of them wrong. To express my own opinion I would again present the opening paragraph of this chapter: "The subject of chronic excessive intestinal putrefaction, as well as the mixed and saccharo-butyric forms, is bound up in such a number of factors that an analysis of the condition as a clinical entity is not easy. In considering the subject we have to deal with the age of the individual, the infection of foods, combinations of foods, the effects of foods upon the bacteriology of the intestinal canal, the bacteriology of the intestinal canal independent of whatever foods are taken, the economic factors pertaining to life, the sociologic conditions under which the individual lives, the status of secretion in the stomach, those poured into the small intestine, those of the intestinal mucous membrane, the status of motility in the stomach, small and large in-

testine, the rate of transit through the intestinal canal, oral and faucial sepsis in relation to a direct infection of the gastro-intestinal canal, various infective conditions such as those locked up in the gall-bladder and appendix, as they may infect the intestinal canal, matters of bands, kinks, adhesions, etc. It will thus be seen that what might be classed as intestinal toxemia is far from a simple condition, even in the individual case."

It can be deduced and believed that chronic excessive intestinal putrefaction and fermentation may be divided into two classes, the primary and the secondary. The primary ones are those which are due to definite infective conditions running chronically in the intestinal content and in the gut wall, while the secondary ones are those which are due to disorders which unfavorably influence the status of affairs in the intestinal canal and bring on the toxemia in a secondary way. Bouchard, Metchnikoff and Herter dealt with a primary cause; Lane dealt with a resulting cause—a stasis which is brought about by interference with the motility due to resorption of bacterial products. In instances where definite obstruction exists due to bands or kinks, we deal with a definite condition for which operation may be indicated, although rarely resulting in the absolute cure of the case, unless obstipation exists. However, Lane should not be given the credit for the benefit of cures from this standpoint, because American surgeons for a long time have been operating upon infected conditions, such as diseased appendices and so on, and have been relieving bands, kinks, adhesions, etc., which have to do with anchoring or binding down the gut. From bacterial changes in primary intestinal toxemia there develops a degeneration in the sympathetic fibers between the muscle planes which may extend into the sympathetic paths extra-enterically even as high as the sympathetic plexuses in the back of the abdomen. When enough degeneration has taken place, dilatation of the viscus which is supplied by these sympathetics occurs, and atony with stasis is the result. Such may cause a sagging of heavy organs which further add an item of delay. Thus, in my opinion, Herter was correct in his opinion as to the original cause, this underlying that of Keith's which in turn can bring about conditions such as Lane described within the abdomen.

Toxins formed in the bowel may conceivably be of four types, first, products of disintegration of foodstuffs by the digestive juices; second, products of disintegration of foodstuffs by bacterial activity; third, the ectotoxin discharged by the intestinal bacteria; and fourth toxins from the dead bodies of bacteria.

In regard to the first type, peptones, proteoses, etc., from proteid digestion are toxic only when introduced directly into the blood or tissues. It has been stated that these could not act as toxic bodies and that if they were introduced, which is not possible since they are not absorbed unchanged by the healthy bowel, only anaphylaxis could take place. It may be well to remember that in the presence of boiling mineral, acid proteins give rise to the following bodies; diamino acids, or hexone bases; lysin, arginin, histidin, ammonia, monamino acids; the aromatic series such as phenylaminopropionic acid or phenylalanin, tyrosin, indolaminopropionic acid or tryptophan, and finally the fatty series such as leucin, glycocoll, aminopropionic acid or alanin, aminovalerianic acid or butylalanin, aspartic acid, and glutamic acid. It therefore must be remembered that products such as peptose, proteoses, etc., never gain entrance into the general economy, but such that may are the albuminous molecules as far as the amino acids and no further. These amino acids may easily gain entrance into the general circulation.

Somewhat the same bodies are formed in laboratory digestion of albuminous substances, with the exception that in this condition the digestion proceeds further in so far that a part of the amino acids is split up into fatty and aromatic bodies such as represented in the aromatic bodies derived from the decomposition of tyrosin and tryptophan, the aromatic oxyacids, phenols, the indoxyls and the skatoxyls. In the simple action of pepsin upon the albuminous molecules we have, in addition to the non-crystalline bodies, ammonia and the diamino acids, monamino acids, also the aromatic series such as is represented in tyrosin, tryptophan and phenylalanin, and the fatty series, leucin, glycocoll, alanin, glutamic acid. The gastric digestion therefore, gives rise to the same products as the action of the acids. The digestion by trypsin gives rise to the same bodies as gastric digestion and the action of boiling acid, the only difference being that trypsin has an action incomparably more powerful, and especially more rapid than pepsin.

In the microbic decomposition of albuminous molecules, in addition to non-crystalline bodies, are ammonia, diamino and monamino acids, which gives rise to the aromatic series, also those of the fatty series, and finally, fatty bodies, such as butyric, caproic, valerianic acids, various ptomaines, and bodies of the aromatic series such as those of the oxyacid group, paraoxyphenylacetic acid, paraoxyphenylpropionic acid; the phenol products, such as phenol and paracresol; the group of indoxyls, indol and skatol; and various gases, such as methane, hydrogen, carbonic acid, sulphureted hydrogen, and methylmercaptan.

From this it will be seen that the microbic digestion gives rise, not only to amino acids, but also to the products of their secondary decomposition; the aromatic and the fatty bodies. The effect of the bacteria upon the albuminous molecules is increased as descent down the intestinal canal is accomplished. It is probable that in health the bacteria of the small intestine do not exercise any unfavorable action upon the albumin, but do cause considerable fermentation of the carbohydrates with the formation of alcohol and fatty acids, such as lactic and succinic. These acids act to inhibit the action of the proteolytic bacteria, but prevent the putrefaction of nitrogenous bodies.

Albuminous putrefaction occurs mostly in the large intestine in which the reaction is more or less alkaline. Hence it is that normally the fermentation of the carbohydrates takes place in the small intestine and that the putrefaction of the nitrogenous bodies occurs in the large. In extreme cases of putrefaction the action of the proteolytic bacteria begins in the lower portion of the small intestine and, according to some of my observations, in a few instances almost in the middle. According to Combe[10] the toxic bodies produced by the disintegration of the albuminous molecules through the influence of microbes is classified as first, the fatty volatile acids; second, the ptomaines and leucomaines; third, the aromatic bodies, and fourth, the gases.

Whenever the fatty acids accumulate in the blood, there results an acid intoxication, or acidosis, which is characterized by a diminution in the alkaline bases of the blood. This acidosis is supposed to occur only when the fatty acids are formed in excess, and this rarely happens except under the influence of the breaking up and fermentation of the ternary bodies, the fats in particular.

It is probable that the fatty volatile acids are not a considerable factor in the production of toxic symptoms. Regarding the leucomaines, little is known excepting that pertaining to the lecithinic leucomaines, such as that found in the yolks of eggs, brains of animals, fish roes, and milk and meat in slight proportions. The intestinal microbic putrefaction in decomposing these foods produces neurin and muscarin, violent poisons. As to how much these substances may affect the body it is not possible to state, because there have been no methods keen enough to examine them so as to recognize them in susceptible amounts, but it may be considered that they are factors of importance at times.

The ptomaines are basic bodies produced by the microbic decomposition of the amino acids. They were discovered by Selmi in

dead bodies, and for that reason he named them ptomaines. Some are harmless, others extremely toxic. Their formation by reduction can take place by the action of the anaërobic bacteria of the intestines.[11]

We possess actual knowledge of several ptomaines belonging to the fatty acid group, ethylendiamin, trimethylendiamin, putrescin, cadaverin, hexamethylendiamin; other ptomaines such as pyridin, parolin and collidin are derived from complex nuclei, and there are a large number of others the composition of which is unknown. All of the above-named substances have been recovered in stool and urine from cases of various conditions. It is a well-known fact that in intestinal putrefaction a relatively large quantity of ptomaines is produced in the intestine. Part of these ptomaines are undoubtedly absorbed, circulate in the blood and may cause toxic phenomena before being eliminated through the kidneys. Unfortunately we do not yet possess any really practical method for detecting the ptomaines or ascertaining their quantity.

That certain aromatic bodies are derived from the putrefaction of nitrogenous food in the intestine is well known. It is also known that a man in a state of inanition may nevertheless produce phenol and indol even when the intestine is empty and contains no food, this probably being produced from the intestinal secretion. While the indols and the phenols are derived from the putrefaction of nitrogenous foods in the intestine it seems to be settled that the oxyacids are derived from the body tissues.

Very probably another aromatic body is derived from the putrefied intestinal juices and most probably from bile. The nearest approach to this is a substance known as chlorocolic acid. It is suggested by Combe[12] that aromatic substances are derived solely from the intestinal putrefaction of the proteins existing in the food and intestinal juices, hence their proportion augments with the intensity of intestinal putrefaction. Other aromatic products are those of the aromatic oxyacids: paraoxyphenylacetic acid, paraoxyphenylpropionic acid, paraoxybenzoic acid, homogentesinic acid, aromatic acids, phenylacetic acid, phenylpropionic acid and hippuric acid. Other toxic bodies are those known as the phenols, the indoxyls, skatol, brenzcatechin, very minutes doses of hydrochinon, alkapton, and the various intestinal gases such as carbonic acid, methane, sulphureted hydrogen, and others.

Against the introduction of these toxic substances when formed in the general body there are certain anti-toxic functions of the organism. These may be described as the character of food, the antitoxic factor of elimination, preventing absorption, the acidity of

the gastric juice, the bile, the pancreatic juice, the reaction of the small intestine, the flora of the large intestine, the defenses of the mucous membrane of the intestine, the liver, the antitoxic glands, and finally the general oxidizing process which takes place as a consequence of metabolism.

It is a well known fact that the intestinal bacteria are not required to carry on the ordinary digestive processes of normal nutrition. This being true, the question may be asked why it is that when so many billions of them are constantly present in the intestinal canal that certain authors can make the statement that they could cause no harm. It is impossible to avoid the entrance of bacteria into the digestive tract. For example, the B. lactis aërogenes, B. coli, B. bifidus, soon adapt themselves to the secretions of this part of the body, and ordinarily hold their own against newcomers. By virtue of their adaptation they are not ordinarily harmful to their host; under certain circumstances they are capable of doing service by giving rise to conditions which discourage the growth of many harmless and harmful species which man cannot readily exclude from his digestive tract. There are many conditions which influence the character and extent of bacterial decomposition in the alimentary tract. Among them are the chemical character of the food, the solubility of the food in the digestive juices, and the volume and composition of these digestive juices.

Intermingled with these factors of food and secretory activity is the influence of aërobic and anaërobic conditions in the digestive tract, and the nature of the bacterial activities which occur there. The initiation of putrefactive decomposition in the digestive tract depends very largely, but probably not exclusively, on the activities of obligate anaërobes, and a portion of the digestive tract is at all times under anaërobic conditions. While the facts all point to the correctness of this view it must nevertheless be remembered that the intestines abound with micro-organisms which are able to attack albumoses and peptones, and to effect the further degradation of the protein molecule, thus entering into a symbiotic action with the strict anaërobes. This symbiosis of anaërobes and aërobes is a biologic phenomenon of consequence in determining the distribution of anaërobic bacterial processes in the digestive tract. Without such symbiotic action, the development of strict anaërobes would be confined to those parts of the digestive tract into which oxygen passes rarely, and then only in small amounts. The large intestine is seldom visited by free oxygen, but it is probably usual in man for the small intestine to contain a little air.

If the stomach exodus is slow, the chances for anaërobic development are good, and hence under these circumstances we frequently find that there are evidences of putrefactive decomposition of food that has been unduly retained in the stomach—namely, the presence of sulphureted hydrogen, mercaptan, butyric acid, etc. On the whole, however, in the average case of intestinal toxemia which we see, no gastric condition can be ascribed as a contributing cause, in fact many gastric conditions are secondary to intestinal toxemias.

While in a few cases of intestinal toxemia it can be proven that putrefaction takes place in the lower end of the small intestine, the usual thing is that it takes place in the colon where anaërobic conditions are more perfect. In the colon the anaërobic conditions are well maintained throughout its entire course and here we find the greatest number of anaërobes and the most pronounced evidence of putrefaction. There is, however, a gradual fall in the number of living bacteria beyond the ileocecal valve so that in the rectum the numbers of cultivable bacteria are very much less than in the ascending colon. There is evidence to prove that under certain conditions the restraint against bacteria which would be inimical to the host may be overcome by errors in diet, depressed general conditions, or alterations in the secretions of the digestive tract, and that thus definite infection by the hemiparasitic bacteria that are present becomes possible, that is, infection of the intestinal content.

Toward adult life great differences exist in the habits of different persons, and these are in a degree reflected in the nature of the bacterial processes of the digestive tract. In adult life there are the individual experiences, new responsibilities, new dangers, an enhanced emotional life, and often a greater proportion of indoor and sedentary habits. The dietary is apt to undergo an alteration in the direction of increased and frequently injudicious liberty and the use of tea and coffee, etc. Also the use of tobacco and alcoholic drinks is either increased or begun. Sooner or later these things lead to slight derangements of digestion which manifest themselves clinically. It is not unusual to find people who are over fifty years of age and apparently robust and well, who possess a slight degree of inability at work, and at times feel tired. With such persons it is not unusual to demonstrate the presence of increased numbers of putrefactive anaërobes in the intestines. These persons, though in good health, are not really robust. A period of sustained hard work is followed by considerable mental and physical fatigue. Dining out and the use of alcoholic drinks are indulgences quickly followed by unpleasant consequences. Exercise out of doors becomes more and more of a necessity, and

the individual becomes conscious of having to live within certain conditions compatible with the performance of his duties.

THE TYPES OF CHRONIC INTESTINAL PUTREFACTION.

According to Herter[13] the variations in the clinical manifestations and pathologic accompaniments of chronic intestinal putrefaction suggest that etiologic conditions vary in different patients. The three types he suggests are: 1. The indolic type of chronic excessive intestinal putrefaction. This is marked by striking indicanuria and probably is due to members of the B. coli group. 2. The saccharo-butyric type of chronic excessive intestinal putrefaction, which seems to be initiated chiefly by the anaërobic forms. In its simplest examples there is very little indol in the gut. 3. A combined type, or cases combining the characteristics of groups 1 and 2.

Indolic Type of Chronic Excessive Intestinal Putrefaction.—In these cases the members of the B. coli group form indol in considerable quantities and often they probably invade the small intestine in large numbers. The bacterial cleavages seem largely to replace normal tryptic digestion. Provisionally we may classify here that type of chronic intestinal indigestion found in marantic children with large abdomens, in the treatment of which children much patience is necessary. At first their digestive processes must be improved. Carbohydrates should be greatly restricted and should be given as rice or Huntley and Palmer biscuits. The milk may be peptonized to promote its earlier absorption. Chicken, beef and mutton are permissible, but they should be finely divided. In a child 5 to 6 years old it may be advisable to give only two meals a day. Considerable benefit seems to follow daily irrigation of the colon, which facilitates the removal of the putrefactive products before they are absorbed. The children should exercise, but should be spared fatigue. They should rest much. Because they stand cold badly, they do best in a mild climate during the winter. Improvement may be possible after several years of rigid regime. The retarded growth, however, is evident even at puberty. Some of these patients seem always susceptible to intestinal disorders, and may never become strikingly robust. The symptoms in the adult are described later on.

The Saccharo-Butyric Type of Chronic Excessive Intestinal Putrefaction.—In this type the seat of the putrefactive process is the large intestine and lower ileum. It is due to the activity of the strictly anaërobic butyric acid producing bacteria. Although our study is not yet exhausted it may confidently be stated that in many cases the B. aërogenes capsulatus and G. P. coccal forms are largely responsible.

With this form may be associated B. putrificus and possibly sometimes the bacillus of malignant edema, although often these are not found in cultures on any of the ordinary media.

The abundance of putrefactive anaërobes, especially the B. aërogenes capsulatus, gives a peculiar character to the intestinal contents. The organisms attack carhohydrates and proteins vigorously and butyric acid is formed from both, together at times with propionic, caproic or valeric acid. These acids are largely responsible for the odor of the stools. From proteids, beside these acids, hydrogen, carbon dioxide and perhaps methane are formed. As a consequence the feces have a low specific gravity and often a decidedly light color. The presence of hydrogen leads to the extensive reduction of bilirubin and other pigments. The Schmidt test with mercury bichloride gives a strong pink color. The stools have an acid reaction, although the acids are neutralized in part by ammonia and other bases formed in the process of putrefaction. It is most probable that the ammonium butyrate acts as an irritant to the gut, causing soft stools or diarrhea. Indol is absent or present in small amounts. Phenol occasionally is found in slight excess. In the urine the ethereal sulphates at times are excessive, although the reason for this is not clear. Mercaptan may be present in the feces as a trace; it also is found in cultures by means of the isatin-sulphuric acid test. It has been noted that as the patient improves the mercaptan becomes less or disappears, but the explanation of this phenomenon is at present unknown.

In nearly all adults the B. aërogenes capsulatus is present in the intestines in small numbers. It is possible that this organism is responsible for repeated slight attacks of intestinal putrefaction, although it may not in these mild cases lessen the duration of life. In some persons in whom a high grade of putrefaction is present, a condition of actual invalidism may be induced and life may be definitely shortened as a consequence of the intoxication.

The presence of ammonium butyrate may give rise to irritation of the intestine and this may be associated with an excessive desquamation of the epithelium, not only in the intestine, but in the mouth and stomach as well. We have evidence of this in the presence of a large number of nuclei in the feces, and it is well recognized that excessive desquamation of the lingual epithelium is associated with digestive disorders. The patients suffer from flatulence. They tolerate carbohydrates and acids badly, and are very liable to attacks of diarrhea after a meal of carbohydrates. Acids may be formed in the mouth of these patients through the influence of anaërobes. This adds to the irritability of the intestine. It is possible that in advanced

cases the B. aërogenes capsulatus may invade the small intestine and there find sugar from which to form butyric acid, etc. After the carbohydrates are thus exhausted, the anaërobic forms in the large intestine set up putrefactive processes in the proteids which exist there.

It is also noteworthy that many patients who suffer from severe intestinal putrefaction are distinctly anemic. The first change in the blood seems to be a decrease in its volume; then the hemoglobin falls somewhat and finally the cells themselves are reduced in number. The grade of anemia varies extremely, from a moderate secondary anemia to the most serious grades of the progressive pernicious form.

The Combined Indolic and Saccharo-Butyric Type of Chronic Excessive Intestinal Putrefaction.—Examples of this type of intestinal putrefaction are common. There are many putrefactive anaërobes in the gut, and also a persistent and well-marked indicanuria, which is but slightly influenced by diet. The nervous symptoms are relatively prominent and appear early. They are, emotional irritability and periods of mental depression; muscular or mental activity soon induces a striking fatigue. Later the blood disturbances may appear. Although these patients have intervals of improvement that continue for months, on the whole the general tendency is downward. They become less robust and recuperate less promptly from every succeeding attack. They may run along for ten or fifteen years in a weak condition, with periods of slow improvement, and finally may present the picture of a pernicious anemia. In others the nervous symptoms increase and the patients may need treatment in a sanitarium or in an asylum for mental disorders.

These various manifestations in different individuals may represent merely a differing reaction to the same poison. Whether the nervous system or the blood shall bear the brunt of the attack is determined by the relative vulnerability of these tissues in that particular individual. It is noticed also that under treatment one group of symptoms may improve quite independently of the other. There is a more rapid advance of invalidism than is the case of either type 1 or type 2 alone. The atrophy of the fat and muscle and the blood changes are present, and perhaps also there are chronic parenchymatous changes in the kidney and liver as a result of the constant poisonous action.

BACTERIAL FACTOR IN INTESTINAL TOXEMIAS.

Distaso[14] divides the organism of the colon into two groups—the non-indologenes and the indologenes, and among the latter finds

those which, acting upon the end products of protein digestion as found in the intestine, elaborate those toxins which, when absorbed, produce symptoms. Notable among the indologenes are those organisms of the B. coli class, which, according to Distaso, present an "ever-present threat against the health of the individual." But nothing occurs until stasis appears, when the colon appears to be one of the degenerating organs and therefore especially susceptible to pathological changes. Putrefaction increases and large quantities of toxins are produced and absorbed. The increase in the content of the colon stretches its walls and induces muscular atrophy. Their weight drags the organ out of place and distorts it so that stagnation is favored and a vicious circle is established. Gradually the character of the flora changes. A smear will show a predominance of Gram-positive bacteria, many of which are cocci. The absolute number of organisms is decreased and there are many spores present. The large intestine appears to serve the sole purpose of furnishing a place in which the residue from digestion is partially dried and stored until it accumulates in sufficient quantity to be removed. The old idea that the bacteria complete the protein digestion begun by the body ferments and that they are necessary for life can be rejected, Recently M. Cohendy[15] has worked with chicks which he hatched from disinfected eggs and kept under aseptic conditions and found that they developed fully as well as control animals and appeared to be normal in every way.

According to Distaso,[16] after working three years, the following were the chief types of organisms which he classified:

Chief Types of Intestinal Flora.

A. Gram-negative Bacilli.

1. B. coli group (B. coli Esch., Durham, B. lactis aërogenes, B. acidi lactici, B. paracoli, B. Proyrud, B. pyocyaneus, etc.)
2. B. variabilis group (anaërobic bacilli with round extremity).
3. B. rigidus group.
4. B. thetaiotomicron group (bacilli very polymorphic, with elliptical form).
5. B. preacutus group (bacilli swollen in the middle and pointed at the extremities).

B. Gram-negative Cocci.

These can be classified under three groups:—

1. Sarcina citrea group, very common in the mouth and in the feces, similar in appearance to the gonococcus.

2. Diplococcus orbiculus group, which are strictly anaërobes.
3. Parvulus group, strictly anaërobic shamm cocci.

C. Gram-positive Bacilli.

1. B. acetogenes group.—The greater number of the Gram-positive bacilli belong to this group. The B. acetogenes is the chief representative, since the B. bifidus and acetogenes are very rare in the feces examined in London.

2. Streptobacillus group.—This microbe always exists in the intestinal flora.

3. Diplob. acuminatus group.—An anaërobic bacillus. It produces butyric acid.

4. B. perfringens group.—An anaërobic microbe. It produces enormous quantities of butyric acid.

5. B. aëdematis maligni group, with the B. sporogenes (Metchnikoff).

6. B. rodella group.—Anaërobic group of very long bacilli.

7. A lemon-shaped bacillus, which is stained by iodine and is described by the author as being a butyric-acid-producing microbe. It has not yet been obtained pure.

D. Gram-positive Cocci.

1. Enterococci group, in chains, less frequently diplococci.

2. Small cocci group.—Are also to be seen corresponding in size to the coccus banani, to the staphylococcus pyogenes, and to the staphylococcus asaccharolyticus.

3. Streptococcus intestinalis group: This group contains the streptococci of Hirsch-Liebmann.

In addition three kinds of spores are to be found.

1. Oval spores, rather large, which may belong to the B. sporogenes and to other

2. Round spores, which may belong to the B. putrificus (Blenstock-Tissier) or to the bacillus of Rodella and to the B. alkaligenes anaërobicus.

3. Very small spores, strongly refractile, belonging to the B. perfringens. These species may be identified by simple microscopical examination.

It is very evident that, as the nitrogenous substances diminish, the bacteria in the intestine become more abundant and the species more numerous. This fact is difficult to explain if we do not consider a factor of great importance—intestinal stasis. When this

is present the conditions which exist in the colon may be compared to those in the test-tube. Another factor in the large intestine is the absence of sugars. Others, and myself have shown that in intestinal putrefaction the large bowel does not contain either albumin or peptone. It contains only derivatives of the latter substance, and the microbic flora present in the large bowel must adapt itself to these surroundings. We find on studying the biology of the organisms that it is the indol-forming organisms which do this and predominate.

From this point of view Distaso has classified the intestinal microbes as follows:

A. Non-Indol-forming Organisms.

1. Amylolytic organisms.—They produce sugar from starch.
2. Saccharolytic organisms.—Divide this group according to the principal acid found in the culture.
   (a) Acetogenic.
   (b) Butyric.
   (c) Lactic-acid-producing, but this group does not exist in the intestinal flora.
3. Asaccharolytic organisms.—These do not touch the sugars with the possible exception of a very slight action on glucose.
4. Gelatinolytic.—These dissolve gelatin, but they do not act upon coagulated albumin.

B. Indol-forming Organisms.

1. Amylolytic.
2. Saccharolytic.
3. Asaccharolytic.
4. Proteolytic.
   (a) Gelatolytic; (b) Peptolytic.

This classification allows us to group the organisms according to their functions—that is, those which produce indol and its congeners and those which have not this property. Moreover, we are enabled to include in definite groups those organisms having similar biological properties with respect to their functions *in vivo*. This grouping leads to the important fact that the organisms of groups having similar biological properties can replace one another, which can be found to occur in changes of nutrition and in pathological conditions. In the human flora there apparently are no organisms which act upon cellulose. In fact, "I do not believe any exist, for on inoculating tubes with filter-paper (Berzelius) in a saline medium the paper is broken down, but not dissolved."[17]

The organisms mentioned above are constantly present in the intestinal flora of adults in the normal state; they are also found in the first stage of intestinal putrefaction. It may be objected to that this flora contains organisms capable of hindering putrefaction, for instance, the B. coli. This, however, is difficult to conceive, since in the colon there are no sugars, and therefore these microbes lack an essential requisite in order to exert their beneficent action. The flora of the normal adult, therefore, is certainly harmful, and, far from defending the organism against infection.

It appears from the peculiar grouping of the microbes found in the colon that the digestive functions as carried out in the large bowel are different from those that take place in the rest of the intestinal tract, because in the colon indol and other bodies belonging to the heterocyclic and aromatic series are elaborated and absorbed. These bodies, when absorbed, severely tax the functions of the body and therefore of the liver. This fact may throw some light on the etiology of diseases of the liver. Moreover, those bodies elaborated by the normal intestinal bacteria are not able to induce peristalsis, in which they differ from those produced by the intestinal flora of breast-fed babies; on the other hand, they may have an inhibitory action on intestinal peristalsis or may even irritate the mucous membrane. These facts lead to the conclusion that the normal flora in adults, beside exerting no beneficent action, may of itself, under certain conditions, be distinctly dangerous.

The amino-acids themselves are non-toxic—indeed they are physiological products of digestion that in the blood stream either are carried to the tissues where they are reconstructed into protein, or if in excess of what is utilized by the body for tissue growth or repair, are deamidized, that is, the nitrogenous portion is split off and converted into urea for elimination leaving the carbohydrate portion of the protein to enter into the carbohydrate metabolism. However, the non-toxic amino-acids under the influence of bacterial growth are capable of yielding basic products, the amines and these are decidedly poisonous. It is to these products formed by the growth of certain bacteria, particularly the bacteria of putrid decomposition, that intestinal toxemia is largely due. Especially is this true of certain ethylamine derivatives, notably of the aromatic amino-acids of protein breakdown. Thus, the well known tyrosine is changed into the highly poisonous parahydroxyphenulethylamine known as tyramine; histidine is changed into histamine; tryptophane into indolethylamine; arginine into agmatine; leucin into isoamulamine; and phenylalanine into phenylethylamine. In each of these instances the

transformation of the aminoacids into the corresponding amine is accompanied by a change in physiological action, a nontoxic substance being transformed into a toxic one. Other changes may occur, but as far as we are at present acquainted with the chemistry of protein decomposition under the conditions of alimentation, it is the formation of amines or amino bases that makes the material toxic.

Since the effect of intestinal toxemia is at first purely functional and becomes organic only after a more or less prolonged period, and since, too, the presence of these toxic substances in a given instance is determined with extreme difficulty, it is not easy to prove directly the relation of cause and effect. Nevertheless, the evidence that justifies the conclusion is in sufficient measures both experimental and clinical.

A phase of this subject was investigated by H. Eppinger and J. Guttmann[18] who set out to determine whether there are substances produced in the intestines which act like hormones on the vegetative nervous system. They note that Eppinger and Hess have clearly defined one among the many so-called intestinal neuroses, namely, vagatonia, whose clinical manifestations result from a stimulation of the vagus center by certain toxic substances produced in the intestine. The severest forms of general vagus stimulation are seen in cases of meat and sausage poisoning, in which there may develop bradycardia, low blood pressure, diarrhea, vomiting, sweating, salivation, eosinophilia, and spasm of the ciliary muscle with miosis. These symptoms, which closely resemble those produced by pilocarpine poisoning, may also arise, though not to as pronounced a degree, in cases of endogenous intestinal intoxication. Particularly in spastic constipation may one frequently observe an analogous syndrome; in which hyperacidity, hypermotility, gastrospasm, bradycardia, and even extrasystole form the chief symptom.

Barger and Dale have succeeded in isolating from ergotoxin, one of the ergot derivatives, a substance B.-imidazolylethylamine, which is apparently identical with the decomposition product of histidin, which in turn results from protein disintegration. Histamine, which is another name given to B.-imidazolylethylamine, produces when injected intravenously into most animals the typical symptoms of stimulation of the autonomous nervous system, namely, a lowering of blood pressure, with a simultaneous rise of pressure in the pulmonary artery, spasm of the bronchial musculature, bradycardia, uterine contractions, and diminution in the salivary and lacrymal secretions. Barger and Dale isolated a second base from ergotoxin, namely, p-oxyphenylethylamine, which may also be obtained by the decom-

position of tyrosin, and is, therefore, named tyramine. This substance resembles suprarenal extract, both in its chemical and in its physiological properties. The above investigators also succeeded in isolating histamine from the normal intestinal mucosa, and suggested that it might be identical with secretin. Other bases have been detected in the human feces, for example, putrescin and cadaverin, both of which are protein decomposition products. Eppinger and Guttmann analyzed the feces of normal individuals and of patients suffering from various intestinal diseases, such as enteritis, proctitis, spastic constipation, paratyphus infection, etc., with respect to the amount of basic nitrogen excreted, and found that there is a marked increase in this excretion in the various pathological conditions of the bowel. The average increase is 100 per cent.

An interesting method of detecting minute quantities of B-imidazolylethylamine was evolved by the above investigators. They found that one or two drops of a 1 to 1000 solution of the above substance, if rubbed into a superficial scratch on the skin of a human being produces in the course of five minutes a typical urticarial wheal. Histamine was the only one in a large series of bases tested that produced this cutaneous reaction. Their next step was to determine whether the feces of patients suffering from chronic urticaria showed an excess in the amount of histamine present. Two typical cases of urticaria showed no such excess. Mellanby and Twost isolated from the intestine a bacillus that converts histidin into imidazolylethylamine. This discovery also tends to support the hypothesis that intestinal intoxication is primarily of bacterial origin.

Herter[19] described a condition which he called indolaceturia which he believed to be due to a definite substance arising in the intestinal tract from the breakdown of tryptophan by bacteria. This substance, chemically, is known as indolacetic acid. The diagnosis is suggested by the addition of concentrated hydrochloric acid to the urine which led to the development of an intense rose color. A study of this color reaction showed it to be identical with the reaction described in 1882 by Nencki and Sieber as the urorosein reaction.[20] In my experience the occurrence of indolacetic acid in the urine is by no means a rare one in toxemia cases and it is usually associated with pathologic conditions of the intestinal tract, such as marked states of catarrhal conditions, generally on the right side of the colon, accompanied by small spots of ulceration; ecchymotic areas, etc. In these instances the fine rose-red color of the urine is quite characteristic. There are urines which on the addition of concentrated hydrochloric acid yield indigo in sufficient amounts to distinctly obscure the rose-

red reaction, and it is necessary in such cases to resort to a special procedure in order to make the distinction certain between the presence of indolacetic acid, or urorosein, and the indoxyl compound.

Indolacetic acid is very slightly soluble in cold water, easily soluble in alcohol and in ether, less easily soluble in chloroform and only slightly soluble in benzol. A solution of indolacetic acid when acidified with hydrochloric acid develops a violet color on heating in the presence of a very small quantity of ferric chloride. It is most probable that the so-called Rosenbach reaction of a purplish color is due to the presence of urorosein and indol at the same time. The product is best tested for by the addition of an equal volume of concentrated hydrochloric acid and the careful addition of a few drops of a 0.2 per cent. solution of potassium nitrite, which brings out the urorosein reaction in an unmistakable manner. Most times, however, the addition of potassium nitrite solution is not necessary, the reaction recurring promptly when the concentrated hydrochloric acid is added to the urine.

An insight into the physiologic and pathologic significance of indolaceturia has been made possible by the researches of Hopkins and Cole[21] and of Ellinger[22] on the constitution of tryptophan. Tryptophan, which may with a high degree of probability be considered as indolamino-propionic acid rather than as skatolaminoacetic acid is set free early in tryptic digestion of proteids. Normally, it is promptly absorbed from the intestine and either burned in the body with the formation of products not at present known or appropriated in some synthetic process. Any obstacle or delay in the absorption of tryptophan favors decomposition by intestinal bacteria. B. coli certainly, and B. bifidus probably, are able to form indolacetic acid from tryptophan. It is certain also that many bacterial symbiotic combinations in the intestine are able to make indolacetic acid from tryptophan. Hence it is not surprising that the decomposition of tryptophan in the intestinal tract can be followed by the appearance of indolacetic acid in the urine. The acid probably appears in the urine as the salt of some common base and does not undergo any pairing with sulphuric acid or glycuronic acid. At present, at least, there is no evidence that such pairing occurs. As both indol and indolacetic acid are derived from tryptophan and from tryptophan only, it is plain that there must be a reciprocal relation between the formation of indolacetic acid and of indol. Thus if the indol production from tryptophan be large, the opportunity for the production of indolacetic acid will be less than would otherwise be the case, and *vice versa,* if indolacetic acid be formed in large amount, there is less opportunity for the pro-

duction of indol, since there is at the present time no evidence that indol is ever derived from indolacetic acid. Only when the tryptophan available in the intestine is abundant as the result of delayed absorption can we expect to get both indol and indolacetic acid in abundance. Clinical experience is wholly in harmony with this view. When indolacetic acid is present in large amounts it is not apt to reach the high grades of intensity sometimes observed when indolaceturia is absent. On the other hand, indicanuria of the most intense type is not apt to be associated with the highest degree of indolaceturia.

Although there is no evidence that indolacetic acid yields indol through the action of the bacteria, it is probable that under special conditions it does give rise to skatol. I am of the opinion with Herter that it is chemically reasonable that skatol should be derived from indolacetic acid. Another product which has been found in urine is the so-called skatol-red. It is thought by some investigators that the urorosein of the urine is identical with skatol-red. Porchet and Hervieux[23] have recently maintained that urorosein and skatol-red are identical. I agree with Herter that this is not true, and cannot be proven even by spectroscopic examination.

Indolacetic acid also arises from the decomposition of proteids in the intestinal tract as can be proven by the clinical observation of cases. This is entirely in harmony with the view that indolacetic acid arises from tryptophan decomposition in the intestinal tract because it can be produced by large excess of feces in cases where there has been no urorosein in the urine before. Thus it appears that excessive feeding of proteins may be concerned with the development of indolacetic acid in the urine and this is a well known fact with indol itself. The condition, however, must be regarded as depending rather on the delayed absorption of tryptophan and suitable bacteria conditions than on the mere over-feeding of proteins. It is probable that the colon bacilli play a part in determining the direction of the main cleavage of tryptophan as to whether urorosein is produced or indol. In my experience the cases that have the largest quantity of urorosein in the urine are those in which the anaërobic bacteria are the prominent bacteria in the intestinal canal. These may be represented singly in instances of gas bacillus or such as the Gram-positive organisms. Occasionally, too, I think one meets with cases of saprophytic organisms of the Gram-positive type that are capable of producing a urorosein urine.

THE RELATION OF BACTERIAL METABOLISM TO CERTAIN FOODS.

It is a well-known fact that perfectly wholesome foods may be prepared and sold in an apparently sound condition and yet contain within themselves the elements of their own destruction in the form of included bacteria, and also bacteria which would be inimical to the human being. Only taking into consideration the question of the effect of such bacteria upon the intestinal canal a number of factors should be thought of. The question of bacteria metabolism, particularly as it is related to proteins and carbohydrates enters largely into the solution of the problems of this nature, since proteins and carbohydrates or their derivatives are the most common and important constituents of foodstuffs in which bacterial development plays a part. In this connection the bacteria is mostly influenced by the presence or absence of utilizable carbohydrate.

It is a well-known fact that B. coli grown in media containing only protein or protein derivatives will produce indol, phenol, hydrogen sulphide, ammonia, and other products indicative of protein decomposition. It is apparent then that the organism of necessity utilizes the protein substances. Putrefaction is the result, because the medium becomes progressively alkaline, foul odors develop, and the resulting products are not only disagreeable to the senses, but are quite unfit for food. This is bacterial putrefaction. The same organism in the same protein medium, containing in addition sugar which the colon bacillus can utilize now produces an entirely different kind of decomposition; in place of the products of putrefaction now appear lactic acid, small amounts of fatty acids, as well as carbon dioxide and hydrogen, which are characteristic of the breakdown of carbohydrate. The reaction now is permanently and progressively acid, the odor not offensive, and the products formed are innocuous and inoffensive. This is bacterial fermentation. It will thus be seen that in the presence of protein and sugar most organisms utilize the sugar in preference to the proteins. In the B. coli for instance this utilization of the carbohydrates instead of the proteins when both are obtainable in the same media is a method of identifying the B. coli characteristically by its sparing action upon the proteins. This is because the carbohydrates are more easily metabolized than proteins, because in the presence of protein they abstract only a small amount of nitrogen for their development. Whenever bacteria are presented simultaneously with protein and utilizable carbohydrate in the same medium the structural needs are largely derived from the proteins, and the fuel requirements from the sugars. Kendall[24] has shown

by determining the increase of ammonia content of media incidental to the growth of bacteria that B. coli produces about 2 milligrams of ammonia above that of uninoculated controls in 100 cubic centimeters of broth containing dextrose and protein. B. coli produces about 20 milligrams of ammonia above the inoculated controls in the same protein broth, but without the dextrose; in sugar-free broth in other words. He states further that the addition of sugar prevented 90 per cent. of the protein breakdown as measured by the ammonia standard in the experiments.

This fact of bacteria sparing the proteins and using the carbohydrates is important in connection with the treatment of putrefactive conditions in the intestinal canal. The object of a diet rich in carbohydrates is twofold—physiologically to provide the patient with a readily assimilable food requiring the minimum amount of digestive energy to prepare it for the tissue needs, and bacteriologically, to shift bacterial metabolism from the destruction of body tissue for their food requirement to the utilization of carbohydrates for at least the major part of their dietary needs. This does not directly result in annihilation of the invading bacteria but it certainly approaches their metabolic reformation. The shifting of metabolism to sugar appears to deprive these microbes of one of their most potent weapons of defense and forces the parasite to act on the defensive, theoretically at least, permitting the host to rally and strengthen his defensive and even his offensive powers earlier in the battle.

While the above is true in the main, there are many cases of intestinal putrefaction in which no changing of the diet to a carbohydrate basis prevents the formation of putrefaction in the content of the intestinal canal. Whenever such is met with in spite of the diet either a pathology is present which has been overlooked or additional methods of treatment are required.

SYMPTOMS OF CHRONIC EXCESSIVE INTESTINAL TOXEMIA.

It is a difficult task to describe the symptoms of intestinal toxemia. On the one hand they may be outlined about as broadly as is the field of medicine, on the other they may be described in detail, both locally in the abdomen, and generally in the body. Then again they may be considered entirely from the laboratory, and conditionally from the X-ray standpoint. Attempt will here be made to classify the symptoms so that a semblance of order will be presented.

Fatigue.—The victim of a long-standing toxemia presents certain general characteristics. In the first place the matter of fatigue is con-

spicuous, and many of these individuals after engaging in moderate amounts of work and mental effort become quite exhausted and perhaps must rest a while before they can recuperate their sense of well-being. This factor of fatigue is quite characteristic in all, and in women it is generally accompanied by more or less headache, pains in the back and perhaps down the limbs, certain neuralgic manifestations together with circulatory ones, such as cold hands and feet, sweating of the palms, and various vasomotor factors. As a class, such persons not only seem fatigued on exertion, but have a constantly running fatigue which brings them closely to more or less inherent devitality in the nervous system. They may have a fairly high hemoglobin content and look quite well, yet, when subjected to any strain or extra effort the result is liable to be disastrous for a number of days. Such individuals are commonly seen among men of importance and those engaged in heavy financial work, or perhaps in teaching. Not uncommonly there may be a mental inability to continue with their efforts after three or four o'clock in the afternoon, and ofttimes such men will drink alcohol in the evening, or perhaps coffee, for the purpose of keeping their well-being until it is time to retire. It is not unusual for them over Sunday to acquire quite a reserve stock of vitality and to engage in work on Monday morning with vim and zest. Perhaps then the feelings of fatigue will not be manifest again until Tuesday afternoon or Wednesday, and toward the end of the week they are quite exhausted, perhaps even the mornings being interfered with so that they cannot conduct their business to their satisfaction. Dizziness is commonly present (Indolic and mixed forms).

Anemia.—Not uncommonly in these individuals chronic anemia is a factor; particularly is this true among the children and the women and those who are more or less housed during the winter time because of sedentary work. This anemia is never pronounced; it is of moderate grade and of the simple type. It is more pronounced where the sulphate partition is high and a condition of indicanuria exists. This is probably due to reduction of the red blood cells from resorbed sulphureted hydrogen gas from the intestine (Indolic forms).

Anorexia.—This is commonly present, such individuals rarely having a sharp appetite. They go along from day to day eating moderate amounts without much desire for food of any kind. They can get along with very little food and generally if they eat a normal amount they are inclined to ascribe to this, the symptoms of distress, and generally begin to eliminate certain articles of diet, so that after a time they are eating less than is required to maintain a fair state of health. At other times their appetite may be considerable and

they eat well for two or three days, and perhaps longer, then suddenly it will drop off again into the chronic condition of loss of appetite and more or less anorexia. (Indolic and mixed forms.)

Insomnia.—Insomnia is quite a characteristic symptom of intestinal putrefaction. It is surprising to see how long many of these individuals continue taking various hypnotic and soporific drugs for the purpose of mastering a persistent insomnia the cause of which has not been corrected or ever handled intelligently. Generally these individuals, who have eaten three meals a day, are considerably more toxic at the end of the day than in the morning, and it is not uncommon for them to lie awake in the early part of the night, getting to sleep when quite exhausted along toward the small hours of the morning, and in the morning they are generally quite sleepy and it is difficult for them to arise. The urine at the time of the insomnia generally contains a high sulphate partition whereas in the morning it may be sulphate low. It is almost as if the toxic bodies absorbed from the intestinal canal stimulate the central nervous system so that normal sleep is not possible. (Indolic and mixed forms.)

Skin.—In addition to the characteristic skin which some of these individuals have, namely a staining particularly manifest under the arms, in the groins, and around the neck, there may be certain forms of skin manifestations of a dermatological order. Eczema in children with intestinal toxemia, and also in adults, is most common. Generally in my experience the most common type is the so-called eczema rubrum. It may be that the eczema itself is not directly caused by the gastro-intestinal condition, but it certainly does aggravate the condition markedly so that the individual is more eczematous than would be the case if they did not have an intestinal toxemia. It is not uncommon when this has been cleared up for an eczema which has been standing for years to entirely disappear, or practically become minimized to such an extent that the individual pays no further attention to it.

Another common finding is the irritative rashes such as are represented in urticaria and erythemia. Recurring urticaria is particularly a symptom of the saccharo-butyric form, but may also exist in the mixed, or even the indolic type. Erythemia that is more or less extensive and quite impracticable to handle by any local application to the skin is not an uncommon accompaniment to these intestinal conditions. Augioneurotic edema may exist.

Another cutaneous eruption caused by toxemia is acne. While acne may be caused by over-heating or eating indigestible foods, indulging in alcohol, smoking, coffee etc. there is no doubt that in many

of the intractible forms the acne is due to the elimination of irritating substances from the intestinal canal in the skin. Less common than acne is acne rosacea which is greatly aggravated by gastro-intestinal disturbance, and not uncommonly in certain neurological individuals is an accompaniment of intestinal toxemia. Lichen planus which usually yields more readily to a vegetable diet, is due, in my opinion, to a chronic indicanuric or chronic putrefactive state of affairs in the intestinal canal rather more than to any other factor that is known. (The three types.)

Nervous System.—There is absolutely no doubt about it that a distinct relationship exists between mental and nervous conditions and these disturbances of the intestinal tract. While certain nervous disorders may cause symptoms in the gastro-intestinal canal, by far the largest majority of these functional disorders are brought about the other way. The commonest of these is the so-called neurasthenia, or nervous exhaustion. While it is true that certain conditions can bring on this disturbance in the nervous system, it is nevertheless true that the commonest result or complication in intestinal toxemia is the so-called neurasthenia. The majority of these people have been treated from a neurological standpoint, sent away, and so on, and many of them get over the major symptoms of the neurasthenia, but have a neurasthenic condition chronically present. This is due to the fact that the intestinal condition has not been corrected. In 4273 cases of intestinal toxemia which I have treated thoroughly, 1126 have had sanitarium experiences for so-called neurasthenia. Thus one can see how commonly exhaustion of the central nervous system is a resulting condition in intestinal poisoning.

The nervous system is almost invariably affected in whole or in part, by chronic intestinal toxemia, and the symptoms in the nervous system may be all that the individual complains of. In every case of such, an examination of the gastro-intestinal tract should be made, and also a search for the cause of the neurasthenia, because it is not normal for an individual of middle age to become neurasthenic without a cause. Of course, this cause may be something other than an intestinal condition but in the majority of instances it cannot be, as is proved by the fact that these individuals do not become well until the intestinal condition has been corrected, and it is not uncommon for people who have been in more or less of a semi-neurasthenic state for years to become entirely well after the intestinal condition has been corrected.

While it probably is not commonly possible for disturbances in the intellectual and psychic spheres to be resulting conditions from

intestinal toxemia, there is no doubt but that a large number of these individuals have a sluggishness of mentality, a dullness and stupidity, a loss of concentration, a loss of memory and incoördination. In the psychic group are irritability, lack of confidence, excessive and useless worry, exaggerated introspection, hypochondriasis, photophobias, depression, melancholic state, impressions, illusions, etc. In a psychasthenic individual who has also an intestinal condition the above mentioned symptoms are invariably intensified.

Von Noorden[25] reports that sensory polyneuritis of a mild grade and pronounced condition of irritatives of vagal disturbance (vagotonia) are commonly due to intestinal toxemia. These he suggests are commonly of enteric origin, the same having been proven by me.

What patients describe as "rheumatism" consisting of pain without any apparent manifestation in joints or muscles, is common among these people. The pains may be transitory or last in the same location for days. They apparently are more myalgic or due to nerve involvement in the muscle planes or perhaps in the sheaths or capsular ligaments about the joints.

Other nervous symptoms often seen present are brachial neuritis, and neuralgias, various painful conditions in the muscles of the back, sciatica, intractible types of headaches, particularly those of the cyclic form, and more or less instability in the vasomotor system such as produces mottled and swollen fingers and conditions resembling chilblains (all forms, but mostly the indolic and mixed).

Mental Diseases.—Ross[26] reports on the examination of urines the following statistics: In ninety-one apparently healthy individuals 7.69 per cent. showed positive urines without the O-agent and 21.37 per cent. positive with O-agent. He claimed that among the insane the indolacetic acid test was most frequent, the results obtained being 43.06 per cent. positive compared to 21.37 positive in apparently healthy individuals. The age factor he believed was not an influential one. The detailed report is on following page: (All forms, but mostly the indolic and mixed.)

SYMPTOMS IN SPECIAL PARTS OF THE BODY DUE TO INTESTINAL TOXEMIA.

Eye Symptoms.—It is not uncommon among these people to find choroiditis, iritis, and various functional disturbances. There have been at least a score of instances of ophthalmologists who have referred cases to me for treatment for some gastro-intestinal condition and only after the intestinal condition had been cleared up did the ocular symptoms disappear. The probabilities are that color

TESTS OF URINES OF INSANE PATIENTS.

Type of Mental Disorder.	No. of Cases.	Test without O-Agent.		Test with O-Agent.	
		No. Positive.	Per Cent. Positive.	No. Positive.	Per Cent. Positive.
Organic brain disease (not differentiated)	7	1	14.29	2	28.58
General paralysis of the Insane	21	2	9.52	4	19.04
Senile dementias	16	2	12.50	10	62.50
Infective exhaustive psychoses	3	1	33.30	3	100.00
Intoxication psychoses (alcohol and morphine)					
Chronic	14	1	7.14	4	28.57
Acute	6	1	16.67	2	33.33
Total	20	2	10.00	6	30.00
Dementia precox group					
Hebephrenic	42	4	9.52	15	35.71
Katatonic	12	5	41.66	10	83.33
Paranoid	18	2	11.11	10	55.55
Not differentiated	102	8	7.84	48	47.06
Total	174	19	10.92	83	47.70
Manic depressive group					
Depressed	16	1	6.25	6	37.50
Excited	17	2	11.76	4	23.52
Remission	2	0	....	0	....
Total	35	3	8.57	10	28.57
Involutional melancholias	5	0	....	2	40.00
Psychoneuroses	2	0	....	2	100.00
Paranoic states	4	1	25.00	3	75.00
Psychopathic personalities	3	0	....	1	33.33
Epileptic psychoses	42	3	7.14	15	35.71
Defective mental development	26	3	11.54	15	57.70
Unclassified cases of insanity*	132	18	13.64	65	49.24
Tuberculosis in insane individuals†	26	3	11.54	10	38.46
Very inactive insane individuals	25	4	16.00	13	52.00
Total insane individuals tested	490	55	11.22	211	43.06

* Old cases which are largely cases of dementia precox undoubtedly.
† These individuals were also included in the different psychosis groups.

blindness and also various spots in the vitreous are largely due to intestinal conditions. (Indolic and mixed forms.)

Asthma.—In the absence of a hereditary history and when a renal and cardiac condition can be excluded not a few cases of chronic essential asthma are due to intestinal toxemia. Some of the most striking results that I have achieved have been in cases of chronic asthma, particularly those that occur in the late part of the winter, from the middle of January on, before the warmer weather sets in.

It is due to the housing up of these individuals and the living under conditions which are conducive to low oxidation and a diet which in the winter time is not as abundant in the fresh varieties of vegetables, etc., as in the summer. (Saccharo-butyric and mixed forms.)

Myocarditis.—In middle age in the absence of syphilis, alcohol or other toxic factors as the cause, and in the presence of a marked status of intestinal putrefaction, this disease may be due to these intestinal states. As a rule, however, when the heart condition is distinct, although a general benefit in health may be brought about by the treatment of the intestinal condition, the heart condition does not clear up. The same may be said of vascular and renal conditions. There is no doubt that intestinal toxemia is a factor of much importance in connection with arteriosclerosis in middle age. When syphilis, lead, alcohol and various other factors can be eliminated a chronic intestinal condition must be taken as the main cause. While with the improvement brought about in the intestinal condition, and perhaps even a complete cure, the stiffness in the vessels always continues, at the same time it does not seem to progress and many of the symptoms of increased pressure, when it exists, and others, are distinctly benefited by treatment of an intestinal state. (Saccharo-butyric and mixed forms.)

Joint Conditions.—Arthritis deformans is, in my opinion, based upon the successful treatment of forty-five cases, largely due to an intestinal condition as the main cause. Some of the most striking successes I have had have been in cases of arthritis deformans. The relapses of the attacks have been stopped and distinct amelioration in the already affected joints has taken place. I do not at all agree with Pemberton in his conclusions that the successful treatment of arthritis deformans is a matter of diet and hygiene, and that the intestinal bacteriology is not an important factor. In my opinion, the most important factor in connection with the cause of this condition is an intestinal toxemia and the treatment in the individual case may not be a matter of diet so much, but may be vaccine entirely. This holds true in both the atrophic and hypertrophic types. Not a few cases of gout are due to the saccharo-butyric form of this condition. (Indolic and mixed forms.)

ABDOMINAL CONDITIONS CAUSED BY INTESTINAL TOXEMIA.

The Esophagus.—Not a few of these individuals complain of substernal distress which on esophagoscopy shows that there is no pathology or congested condition of the esophageal mucous membrane. In

my opinion various substernal distresses, probably esophageal in origin, are due to reversed peristalsis taking place through the cardia and stomach of individuals who have more or less intestinal toxemia. (All forms.)

Fig. 46.—Section through normal ganglion of Auerbach plexus, showing communicating fibers. (*Golgi* method.)

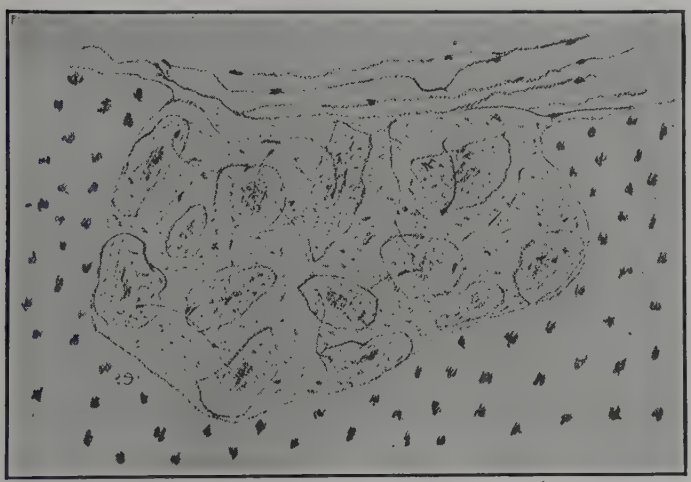


Fig. 47.—Section through a ganglion of the Auerbach plexus in a case of chronic intestinal toxemia, showing deformity and contraction of cell bodies with granular degeneration of cytoplasm.

Hyperacidity and Hypersecretion.—The textbooks on diseases of the stomach, for a long time have described these conditions as entities without giving any causes beyond indiscretion in diet and drinking, and ulcer. The truth is that by far the largest number of cases of hyperacidity are due to chronic intestinal toxemia, generally of saccharo-butyric type. It is not uncommon to find a hyperacidity

existing at least for a while in the early stage of putrefaction in the intestine. But generally the picture in the putrefaction case is one of a lowered amount of secretion all the way to an achylia, which is due to an atrophic gastritis, the commonest cause of atrophic gastritis we have.

Both hypermotility such as is represented in an irritative condition of affairs, perhaps a status of vagotonia, and also a condition of hypomotility, where there is a slight slowing in the exodus power of the stomach, are present in cases of intestinal toxemia. Most of the instances, however, are those of dilatation plus a diminution in the height and number of the peristaltic waves that run in the stomach—considering eight to ten to the minute as normal. Such stomachs do not manifest a distinct stasis and generally are empty of the barium meal in six hours. To note the slight degree of exodus power, it is necessary to use the test-meal method and extract every fourth, fifth and sixth hours, and perhaps examine wash water for food remnants. (All forms.)

Atony.—Those states of loss of tone in the musculature of the stomach as well as diminution of peristaltic power, in my opinion, are due to an effect upon the Auerbach and Meissner plexuses by resorbed intestinal toxins. Such are not as manifest in stomach conditions as they are in conditions of the colon. In the colon careful sectioning and staining of the nerve tissues between the muscle planes and on the entering side of the external plane show distinctly that in intestinal toxemia a granular degeneration of the cytoplasm may take place with the shrinking of the ganglionic-cells, and these cells are presided over by the splanchnic which have to do with maintaining tone within the gut. Such degeneration and shrinking causes a diminution and a certain amount of cut-off of the batteries from the rear, and dilatation is the result. Still it can be said without a question that insofar as the stomach is concerned, an intestinal toxemia is capable of bringing about marked states of atony. The same may also be said in some cases of pylorospasm. Recently it has been suggested that pylorospasm is due to a chronic colitis, as well as a referred condition from disease of the appendix, gall-bladder, etc. In my opinion a functional pylorospasm can take place, the type being of moderate or slight degree, in cases of intestinal toxemia, and it must be remembered that chronic colitis is merely a resulting condition from a disturbance in the intestinal canal of the kinds that I am describing. (All forms.)

Hyperesthesia Gastrica.—By far the largest number of cases of hyperesthesia gastrica are due to indiscretions in diet, but not a few

occur as a result of a long-standing intestinal toxemia, and these often are best treated by considering them as intestinal in origin. (Saccharobutyric and mixed forms.)

Pyloritis.—A condition hitherto not described in which the symptoms may be marked and which may simulate the presence of an ulcer is an inflammatory disorder of the distal one-third of the stomach, generally a primary congestion which may even go on to an inflammation and more or less organization of tissues, and which I have designated as pyloritis. These instances, in my opinion, are due to disturbances of the internal secretion partly and local infection in achylic stomachs, but mainly to intestinal toxemia, and they may be so bad as to require operation for stenosis and other reasons. (All forms.)

Ileal Stasis.—By far the largest number of cases of putrefaction in the intestine have an ileal stasis. These represent just as definite a degree of stasis as when there is pathology in the region of the ileocecal valve. This stasis which seems manifest in the six hour plate by a Roentgenographic examination may not be a stasis after all. Recent investigations of mine prove the accuracy of Alvarez[27] that the anti-peristalsis in the right colon may be so active as to cause regurgitation or more or less stasis in the lower end of the intestine. I think probably the stasis which is apparent on the plate is due to the anti-peristalsis in the right colon, perhaps a spastic condition of the transverse descending colon, due to the intestinal toxemia, the whole picture being a general slowing up of the transit through from the lower end of the small intestine to the anus. (Indolic and mixed forms.)

Appendix.—It must be perfectly logical to anyone to consider that the large number of cases of acute and chronic disease of the appendix must have its origin in the bacteriology of the intestinal canal. My belief is that the main cause of chronic appendicitis is an intestinal toxemia, and this explains why it is that often after appendices are removed the symptoms continue just as before, because removal of the appendix will not cure intestinal toxemia. There is a feeling nowadays that in an instance of intestinal toxemia and diseased appendix, the removal of the appendix will have a beneficial effect upon the intestinal toxemia. This is true in only the minority of instances—the great majority of cases are the other way—namely, that the intestinal toxemia is primary and not removed by operation, and the removal of the appendix merely removes a resulting condition and not the actual cause. (All forms.)

Megacecum.—This condition in the idiopathic type commonly is a resulting condition of intestinal toxemia. It is brought about, as mentioned before, by a resorption of toxic bodies from the interior of the gut and a gradual degeneration and shrinking of the cytoplasm of the cells in the Meissner and Auerbach plexuses in the right colon. Generally there is more or less of a catarrhal condition present at the same time—a so-called right sided colitis. (All forms.)

Chronic Colitis.—Chronic colitis is often a resulting condition from an intestinal toxemia of long standing. Generally there is disease of the mucous membrane of the right side of the colon with perhaps more or less atrophic changes in the lower end of the colon and sigmoid as well. The present standard of medicine of treating cases of chronic colitis with irrigation and efforts at dieting without a knowledge of the bacteriology of the intestinal canal is far from scientific, and the results brought about are usually not practical or substantial. About all that irrigation can do is to cause an emptying of the colon. Some benefit may come in that way but usually the primary condition is not cured. Generally there is more or less dilatation of the colon in these cases, although it is not always found on X-ray examination. Usually, there is a distinct change in the mucous membrane to the extent of a hypertrophic inflammation or an atrophic destruction, dry in type. The hypertrophic type is usually accompanied by more or less spasm, which is commonly expressed as spastic constipation since constipation usually exists in these cases. The atrophic form has generally a dilated gut with a dry mucous membrane. A condition of constipation exists here too, but the constipation is due to the dryness of the mucous membrane plus the atony. All there is to chronic colitis, spastic constipation, atony of the colon, colonic stasis, etc., is merely the story of chronic intestinal toxemia of long-standing and not treated in the proper way. The same may be said of hypertrophic and atrophic proctitis. (Saccharobutyric and mixed forms.)

Adhesions.—It is not uncommon to find pericolonic adhesions in these cases. Adhesions may be found in the right colon or in the descending, perhaps only in the region of the brim of the pelvis on the left side. They are due to migration of bacteria through the mucous membrane and walls of the gut, and gaining the peritoneal surface, causing a plastic and adhesive form of a low degree of peritonitis with adhesion formation, the original condition being intestinal toxemia. Some time ago I drew attention to a bacterium which is most active in its ability to migrate through the walls of the gut and cause adhesions on the peritoneal surface[28] (B. adhesiofor-

mans). This bacterium, however, is not the only one capable of producing adhesions. The condition is brought about, firstly, by an intestinal toxemia, then the production of a chronic colitis and the breaking down of the local resistance of the mucous membrane, the migration of bacteria through the mucous membrane and gaining the peritoneal surface with resulting pericolonic adhesions. There are various forms of toxemia that are capable of doing this in susceptible individuals. Dry sigmoiditis is quite common. (All forms.)

Ptosis.—There is no doubt but that intestinal toxemia has an important bearing in connection with ptosis. Cases of ptosis plus toxemia must be divided into two groups—those with secondary and those with primary intestinal toxemia. In those that have a secondary intestinal toxemia, due to the ptosis, the treatment is that of ptosis, after which the intestinal toxemia will disappear, generally also with such stasis as exists. One-half of all cases of ptosis however have a primary intestinal toxemia. To benefit the ptosis and relieve the symptoms by the incorporation of all of the methods of treatment that are known for ptosis benefits for a short time only, with a resumption of the symptoms later on. This is because the original primary toxemia has been left, eventually again causing symptoms in the abdomen. This explains why it is that some of the cases of ptosis are well for a while after treatment, only to recur. In 2400 cases of ptosis that I have had in private practice 48 per cent. have had a primary intestinal toxemia which required three or four months of steady work after the symptoms due to the ptosis had been benefited. (Indolic and mixed forms.)

Gall-Bladder Conditions.—It is a well-known fact that in some individuals, particularly those who have intestinal toxemia, the bacteria may reach the general circulation, in which instance the liver may act as an organ of elimination, the bacteria gaining the bile and collecting in the gall-bladder may infect it with the production of cholecystitis all the way from the strawberry type to that of the fibrous form, and when the gall-bladder is enough diseased as in individuals who have a cholesteremia there can occur a production of gall-stones. Unless such a toxemia is corrected after an operation on the gall-bladder symptoms are liable to be re-established in a year or so. (All forms.)

It may here be mentioned that intestinal toxemia is an active cause of cirrhosis of the liver. It is a well-known fact that many alcoholics do not get cirrhosis of the liver, and that others who drink do. It is also known that cirrhosis of the liver is not an uncommon finding in the operating-room with people who have drunk no alcohol

at all. In investigating this subject I have come to the conclusion that it is not the alcohol that causes cirrhosis of the liver, but the drinking of alcohol and malt fluids brings about a change in the bacteriology of the small intestine and this change is capable of producing cirrhosis of the liver, due to resorption of toxins. People who have not drunk alcohol may nevertheless have this form of intestinal toxemia, and in those instances cirrhosis of the liver may and commonly does exist. (Saccharo-butyric form.)

LABORATORY EXAMINATION.

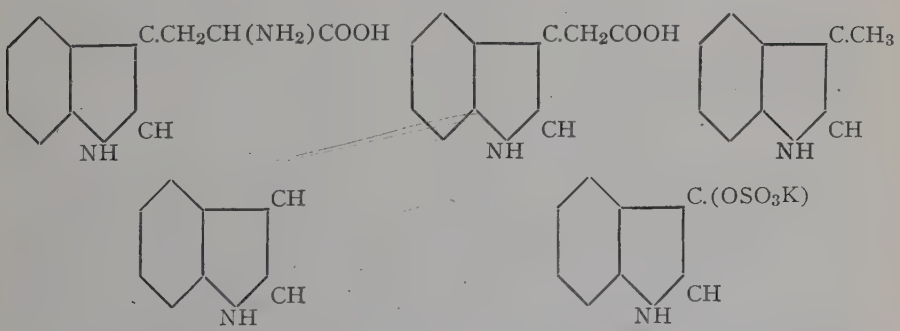
The clinical symptoms being suggestive of intestinal toxemia a complete laboratory and Roentgenographical examination is necessary to complete the diagnosis and to further examine the case in the way of detailing facts upon which it will be possible to make a super-diagnosis as to whether the toxemia is primary or secondary. Since the taking of a barium or bismuth meal interferes with the chemism of the gastro-intestinal canal, it is my custom to resort to laboratory procedures first, doing the X-ray work after it. For this purpose the patient is put upon the test diet that I employ; mentioned in the fifth chapter in connection with testing of intestinal functions and the examination of feces and urine. When the specimens of stool and urine have been received the patient reports with a double test-meal, namely, a Riegel meal with prunes and raisins taken 6 hours before the extraction of an Ewald meal taken 60 minutes before the patient is in the laboratory. This extraction of the Ewald meal is timed at about noon so that promptly after its extraction the patient is sent into the X-ray department and the Roentgenographic examination begun. The whole diagnosis takes the best part of a week, and when the case is examined in this way four different diagnoses are made—first, a diagnosis is made from the history; second, the physical examination; third, the laboratory, and fourth, from the X-ray examination. With all this data on hand one sits down with the facts and studies them in a dispassionate way to figure how the various findings are correlated. Only by this thorough means of examination is it possible to do satisfactory work in connection with intestinal toxemia as well as in the whole realm of abdominal diagnosis.

The Urine.—After measuring the entire quantity of urine, a macroscopic examination is made as to its character. Acidity is tested for and figured on a basis compared to normal. The method I use is the ordinary Tycos acidimeter in which test a decinormal solution of sodium hydrate is used as a titrator, phenolphthalein being used as the indicator. Usually high values of acidification are observed, partic-

ularly in cases of saccharo-butyric fermentation; in cases in which the putrefaction is marked the urine may be normal, or even subnormal in acidity.

Tests are made for albumin, special note being paid to the matter of small quantities. It is not unusual to find these individuals having traces, or sometimes more, of albumin in the urine. Generally, unless the kidneys are involved, there are no casts, the quantity of leucocytes is low, and there are no evidences of renal degeneration. The presence of small quantities of albumin are very important because it is not unusual that when the intestinal condition is cleared up the slight albuminuria disappears. Tests are also made for the presence of glucose and the other sugar bodies. One not uncommonly finds slight glucose, with the presence of albumin. Most often the quantity of glucose is too small to estimate, just giving a slight change with the Benedict solution or perhaps with Fehling's which is more sensitive, particularly when standing over night.

Since indol and indolacetic acid are derived from tryptophan and tryptophan is abundant in animal tissues and in the daily diet of man, and since these substances are well-known toxic substances which in normal metabolism are made non-toxic by their transformation into ethereal sulphate or may not be, these two bodies are next examined for. The close association which exists between tryptophan on the one hand and these ethereal sulphates on the other has been well shown by Ross[29] in the following formula, the substances and their significances having already been mentioned.



It is sufficient to say here that only infinitesimal quantities of indol are to be found in normal urines, and indolacetic acid not at all, and various types of toxemia putrefaction cases are characterized by a large quantity of indol, the saccharo-butyric by a large quantity of indolacetic acid, and the mixed forms by both being present at the same time. When large quantities of these substances are present

intestinal toxemia must be considered as strongly suggested. The presence of skatol may be examined for by the Jaffé test, the Rosenbach or the amylic alcohol test. We now proceed with a more detailed examination of the urine.

While an accurate chemical method of weighing the total solids of the urine is too tedious for general clinical work, for the work in connection with gastro-intestinal conditions it should be attempted. The normal average weight of solids in urines is 65 grams. While the specific gravity is a pretty fair index of the total solids excreted by the urine it still does not serve to accurate purpose. The Trapp or Bird's methods are employed in estimating total solids.

The amount of phosphoric acid excreted is carefully measured, normally it being from 2.3 to 3.5 grams, an average being about 2.8 grams. While of itself the excretion of phosphoric acid is not of great significance in connection with diagnosis, at the same time it largely indexes the character of the diet, matters of exercise, oxidation of the individual, etc. These may be important factors in connection with the cause of toxemia, or when corrected have to do with the amelioration of the symptoms. At other times, however, the presence of large or increased amounts of phosphoric acid may be seen in conditions where there is considerable disturbance in the central nervous system particularly in cases of neurasthenia and debility. The presence of large amounts of phosphates may be taken generally as an index of intestinal disturbance. It is not uncommon in these people to find what has been termed a phosphoturia. For this reason it is important to make a careful estimation of the phosphates. The procedure employed by me is a separate estimation of the earthy and alkaline phosphates. This need not be described in detail here.

The Sulphates.—The major portion of the sulphates appearing in the urine are derived from the food and comprise the simple mineral sulphate, sodium and potassium, only a small proportion existing in organic combination as the ethereal or conjugate sulphates. The predominating conjugate sulphates are phenol potassium sulphate, indoxyl potassium sulphate, (indican) and skatol potassium sulphate (skatol). The mineral sulphates comprise about nine-tenths of the total sulphates in the urine, and we therefore use a measure describing the quantity of ethereal sulphate as 9 to 1, 10 to 1, 11 to 1, or the other way, namely, an increase in the ethereal sulphate such as 9 to 2, 9 to 3, etc. An accurate method of quantitative determination of indican has been described by Askenstedt.[30] Usually, however, the chemical test mentioned above serves to good enough purpose, and an accurate determination of the sulphate partition is made by means of the Folin

method for quantitative estimation of total sulphates and also the Folin method for quantitative estimation of the conjugate sulphates. This quantitative method for sulphate estimation is most important and must always be engaged in while making a diagnosis of intestinal toxemia. The diet being known gives a standard in that to go by, and one can easily figure when increased amounts of conjugate sulphates have been formed in the body. All that is necessary then is to exclude the presence of collections of fluid, pus, etc. which might cause an increase of conjugate sulphates in the urine. These being excluded, the intestinal canal must be looked upon as the source of supply.

Under ordinary conditions a urea estimation serves for the examination of protein metabolism, and the index of nitrogenous equilibrium and elimination. The method for this is well known. The examination of the blood for non-protein nitrogen and urea, may be called for in some cases.

The normal proportion of uric acid to urea is as 1 to 45. In health it exists in solution as potassium urate. A healthy adult excretes 2 to 1.0 grams of uric acid in the course of twenty-four hours. The amount increases physiologically with increased ingestion of food, and pathologically with increased nitrogen metabolism in about the same proportions as urea. In my experience, however, there are instances in which the uric acid content does not correspond to the content of urea, and for that reason I use a quantitative estimation of uric acid in connection with this subject. While one may by studying a centrifuged specimen of urine gain a relatively accurate idea of the quantity of free uric acid there is in the urine, at the same time the urates are in solution and it is necessary to estimate the total uric acid output. The quantitative test for the estimation of uric acid is the Ruhemann's test which with the uricometer serves to accurate purpose.

Oxalic Acid.—The next important body is the estimation of oxalic acid which appears in normal urines only in very small amounts and in combination with calcium as calcium oxalate. The characteristic appearance of these crystals under the microscope is well known and generally the examination of the sedimentary field in a centrifuged specimen of urine is all that is necessary to estimate whether oxalic acid is present in increased amounts or not. Generally when it is in excess the crystals appear in the urine. In many cases of intestinal toxemia the oxalic acid output is very much increased, therefore it is necessary to quantitatively estimate the oxalic acid output. In my laboratory the Baldwin method is employed. Sometimes, however, after studying the sedimentary field when the quantity of oxalic acid

crystals are not in marked amounts a simple method, such as the precipitation of calcium oxalate by alcohol, centrifuging and then studying the field again serves sufficiently for clinical purposes.

Ammonia.—Ammonia partitions are made in all instances which show the presence of sugar, the diagnosis here being along the lines of estimating if possible whether the intestinal toxemia has had some effect upon the pancreas, or whether perhaps a condition of true diabetes mellitus exists, the treatment of which may be very different from that employed in the care of an intestinal toxemia, although sometimes in cases of the saccharo-butyric type the diet would be practically the same for both. Tests for acetone and diacetic acid are also required in instances of diabetic urine.

Bile Pigments.—It is sometimes important to test for the presence of bile acids. Usually bile in urine is manifest by macroscopic examination. The test for bile acids employed by me are the so-called Hays and Oliver test.

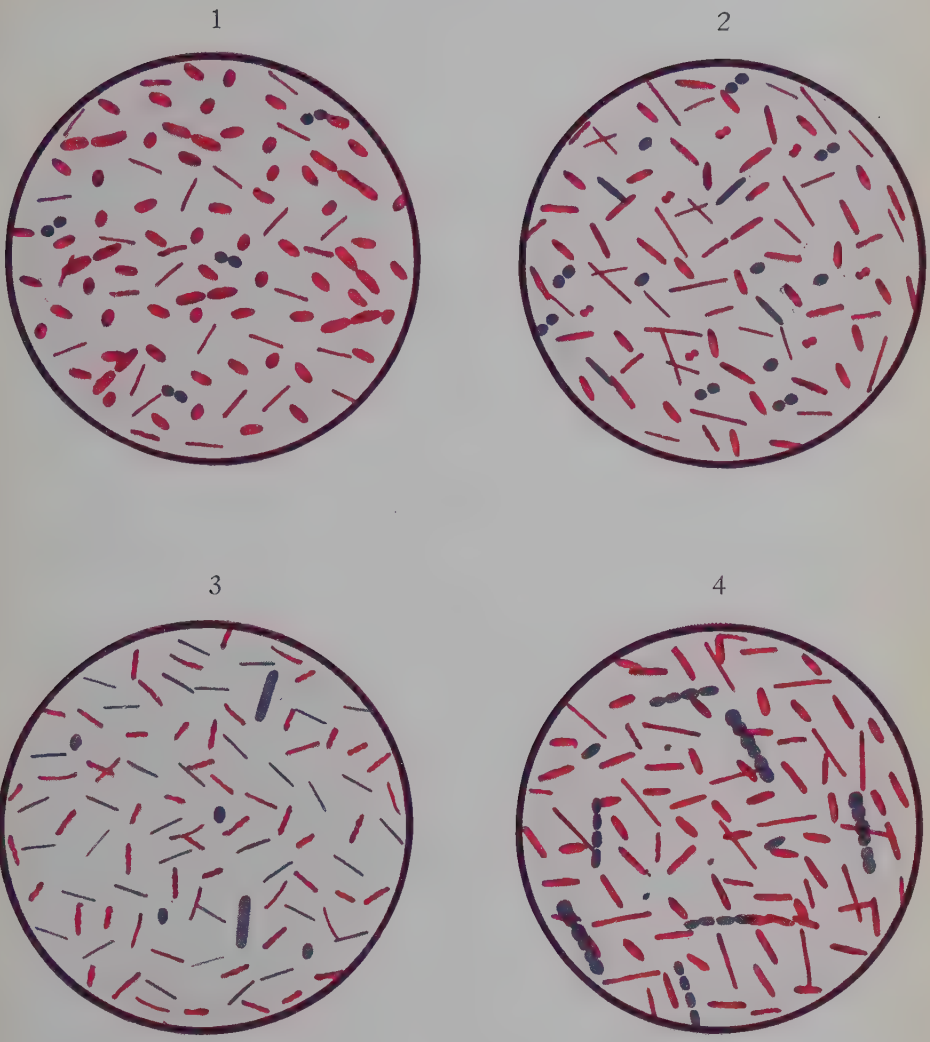
Tests for Functional Renal Capacity.—When considerable amounts of albumin or considerable numbers of casts are present tests are conducted for functional renal capacity. The one most depended upon is the technique of Geraghty and Rowntree, which in my opinion is the best test for functional ability of the kidneys that we have.

Tests for Function of the Liver.—Occasionally it is necessary to employ the test devised by Ehrlich or that of Hirose.[31] The urobilin test, which need not be described, should be mentioned as being of much value in some of these cases.

Examination of the Feces in Intestinal Toxemia.—All that has been mentioned in Chapter V on the examination of feces holds good here. A most exhaustive examination is always entered upon, taking the feces through all of the tests that are known and careful observations made until the very last one. The number of stools, the consistency and form, the odor, color, tests for blood and various food detritus, the quantity of mucus and mucus cylinders, biliary and intestinal concretion, crystalline substances, the bacteriology, and all that pertains to animal parasitology, and the reaction, all are studied. I need not go over this entire field again, it having been presented before, and such that has not been presented can be found in connection with the various well-known works on clinical diagnosis.

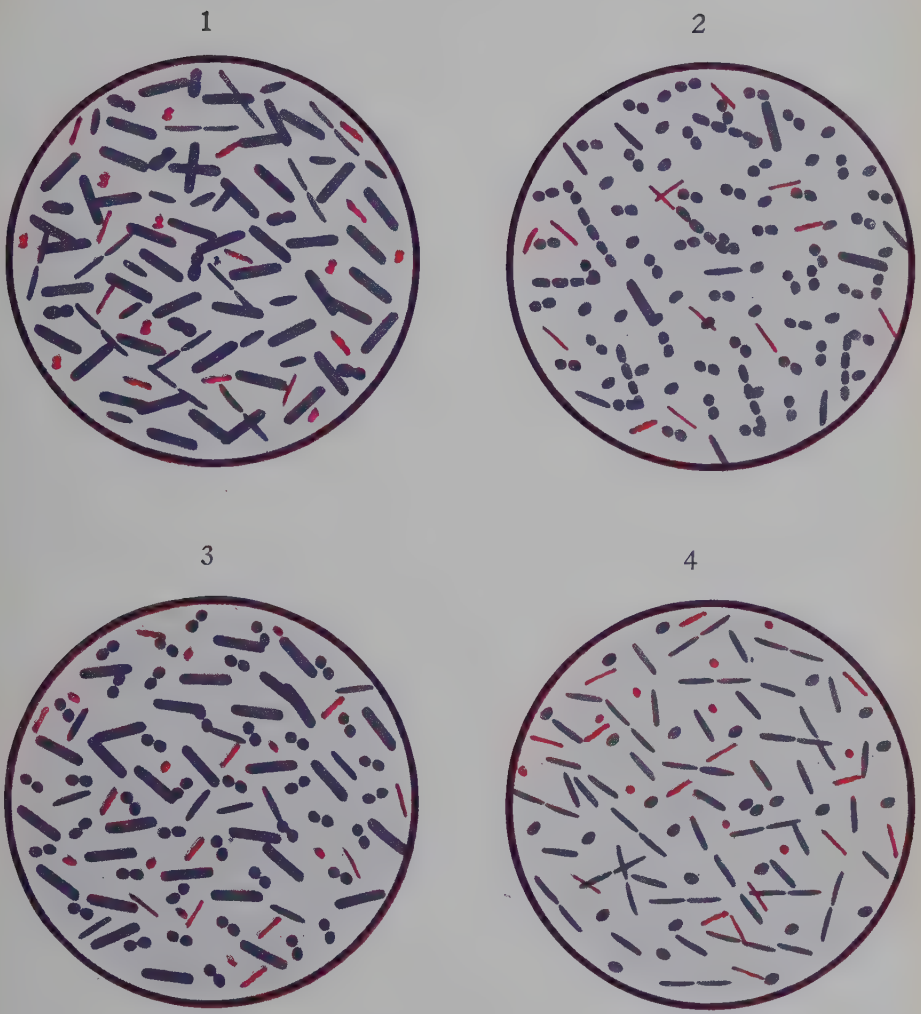
In connection with the fermentation test with the authors' gasometer, or that devised by Strassberger, careful studies of sedimentary fields of inoculated media grown under aërobic and anaërobic conditions are engaged in. The media employed are plain bouillon, 2

PLATE X



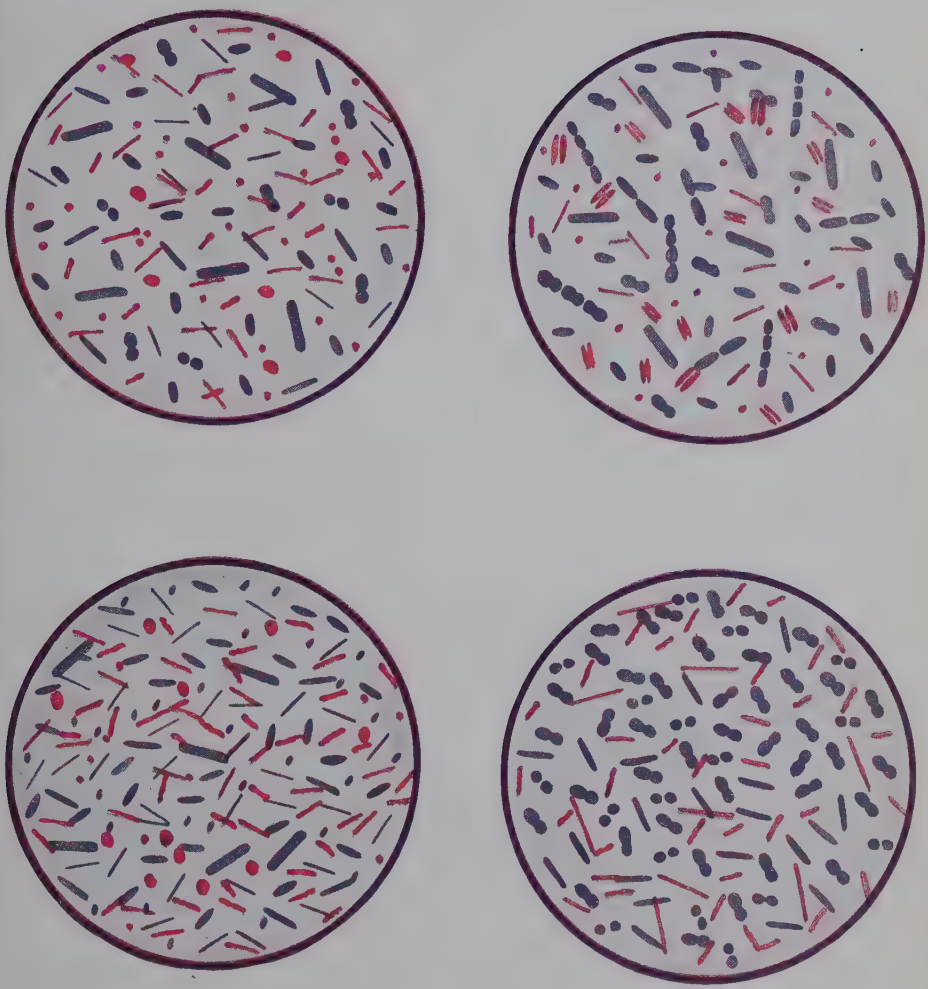
Indolic form of intestinal putrefaction, showing the prominence of the Gram-negative organisms. Fig. 1, A high infection of organisms of the colon group. Fig. 2, A coli infection developed directly from the saccharo-butyric. Fig. 3, Bacillus putrificus plus the coli infection. Fig. 4, The coli infection plus a saprophytic (rare).

PLATE XI



Saccharo-butyric form of intestinal putrefaction, showing the prominence of the Gram-positive organisms. Fig. 1, A Bacillus aërogenes capsulatus infection. Fig. 2, A diplococci infection. Fig. 3, A mixed Bacillus aërogenes capsulatus and Gram-positive diplococci infection. Fig. 4, A small Bacillus aërogenes capsulatus and single Gram-positive coccal infection.

PLATE XII



Mixed form of intestinal putrefaction. Bacterial pictures of feces of four cases.

per cent, dextrose bouillon, blood serum media, the method of Cohendi, and last is that in which extracts of human feces fluid is used.

The different sediments are examined from day to day and careful note made of the change in the bacteriology by methods employed to estimate the bacteriology as it may occur as significant in the course of the examination. Altogether, the study of the sedimentary fields is a matter of six or seven days careful examination. Much significance is placed upon the bacteria which exist at the end of the observation—that is after the second day, these being figured as the more important in connection with the subject. Careful studies are made of symbiotic cliques as they occur in the sedimentary field in an abnormal way as the days go on. The Gram differential method of staining is employed from the first examination of the feces until the last examination of the sedimentary field has been made. This method serves the best purpose in all of the intestinal bacteriological work.

It is this study of the bacteriology which is the one most important factor in connection with intestinal toxemia. Without a careful study of the bacteriology, it is not possible to decide what is the proper vaccine to use. As one gains in experience in the study of the sedimentary fields, it is surprising to see, after the use of different media, how much information well worth the while can be gained. It is almost impossible to detail this in a definite way. Special knowledge of intestinal bacteriology is important to have and a reading knowledge can be culled from the various works upon the subject. No diagnosis of an intestinal toxemia is complete without a careful bacteriological observation of the stools, and also no treatment for intestinal toxemia is complete unless the vaccine method employed is logical and definitely indicated in the case. Unfortunately this entails time, good laboratory work, special knowledge and experience, but to do justice to the subject and the patient it must be done.

THE ROENTGENOGRAPHICAL EXAMINATION.

During the time that examinations of the stool and urine are being conducted in the laboratory the patient should be examined in the X-ray department. All that pertains to the knowledge and advances that have been made in this work is important in connection with intestinal toxemia, particularly in regard to the separation of the secondary from the primary types, and the noting of conditions resulting from a long-standing intestinal toxemia wherein the condition itself, such as a chronic appendix, marked degree of stasis, gall-

stones, etc. could exist as a clinical entity in the production of symptoms. But it must be remembered, even if it is against the teaching upon and the feelings in the subject today, that these conditions are biological and not dynamic entirely. This is true from a diagnostic standpoint, that of cause and effect, and wisdom in the matter of treatment.

Gastric stasis of a distinct degree is generally due to a pathology in the stomach or at the exit of the stomach. It may be due to a pylorospasm which is referred from the appendix region, or perhaps one which is referred from the gall-bladder. Great care must be taken in differentiating the functional types of the pylorospasm and those which are due to a distinct pathology generally existing elsewhere in the abdomen. This entails a careful examination of the gall-bladder and appendix. When pathology at these sites can be excluded and pyloric stenosis of an organic degree does not exist, we must then conclude (if the pylorospasm is moderate in extent) that it is a referred condition possibly from the colon. When after careful work it is assumed to be due to the colon, and the findings in the laboratory would suggest this, the case may be considered as functional pylorospasm. It must be remembered that as a rule distinct 6-hour stagnation of the stomach is due to a definite pathology. There are, however, a few cases in which an atony or ptosis of marked extents may exist which will leave a shadow of bismuth or barium in the stomach, there being no actual pathology in the abdomen if the case is operated upon. But these cases are rare and had best not be considered in connection with the subject. The rule is that where definite stagnation exists pathology should be considered present.

Ileal stasis, however, may be entirely due to an intestinal toxemia. In fact, the vast majority of cases are of that sort. A pathology may exist in the appendix region and there may even be an incompetent ileocecal valve, but the vast majority of cases of ileal stasis are functional in type and due to intestinal toxemia, and are best treated when handled along those lines, namely, medically instead of surgically.

Incompetency of the ileocecal valve is usually a resulting condition of a long-standing intestinal toxemia, therefore the vast majority of cases do not require surgical repair but require medical treatment for the intestinal toxemia instead.

Dilatation and catarrhal conditions of the right side of the colon, comprising the cecum and ascending colon, are particularly resulting states due to intestinal toxemia and should be considered as such, and

medical primarily. The mortality in surgery is too high and the results from surgery not sufficient to warrant it as a definite method of procedure. We then come to general dilatation of the colon, the cases of chronic colitis, those which have a spastic condition of the transverse and descending colon and constipation, or perhaps, on the other hand, atony instead, with dilatation and absence of peristaltic waves, these are distinctly medical conditions as well.

Various factors pertaining to adhesions such as adhesions in the region of the ileocecal valve, ascending colon, hepatic flexure, as well as in the transverse and descending colon, and in the pelvic colon of the left side, these are generally resulting conditions from an intestinal toxemia and if they do not cause an anchoring of the gut at that location interfering with the peristalsis, perhaps a bridging and a distinct stenosis, they should be considered as medical conditions. It is not uncommon that after handling a case of intestinal toxemia with adhesions along medical lines for a length of time that the adhesions either tend to disappear or cease to be causative in producing symptoms.

Colonic Stasis.—Colonic stasis, like ileal stasis, is distinctly a resulting condition from intestinal toxemia and should be considered as a medical proposition, unless the stasis is so marked that practically what is equivalent to a distinct obstruction exists, and this is rare. The question then would come up as to whether the obstruction was conducive to producing the toxemia or whether the obstruction was not sufficient to interfere with the transit through, considering the factors of changing the character of the food and other things so as to encourage its progress.

The symptoms of ptosis are commonly a resulting condition from a long-standing intestinal toxemia. About half of the cases have ptosis in a primary way and are toxemic in a secondary sense. These are best handled by the treatment of ptosis and when benefit has been brought about, the toxemic symptoms disappear, or at least are ameliorated. Ofttimes then they improve enough so that there are no subjective symptoms in the case. On the other hand, those which have a primary toxemia are not substantially improved by long-standing treatment of the ptosis. After the ptosis has been benefited by the ambulatory or rest-in-bed and food treatment, a treatment for the intestinal toxemia should be instituted. This generally means a change of diet, together with the necessary vaccines for four or five months after the ptosis has been attended to. No one is warranted to say that a cure has been accomplished in ptosis unless a careful examination of the stools and urine have been made for the purpose of noting whether

a primary toxemia exists, and then a cure of that toxemia. Of course in these cases there is often an inherent debility which may render the digestive tract decompensated again in the way of producing symptoms from the ptosis. That is another matter, but in the main the toxemia must be benefited or the ptosis symptoms will return in a short time, generally inside of a year.

Gastric and Duodenal Ulcer.—It is my belief that a large number of cases of gastroduodenal ulcer are secondary to intestinal toxemia. Generally in these instances there is also a pathological appendix. The whole canal then has become more or less changed in the way of local pathology such as has been described. This brings one to speak of the results of medical treatment of ulcer. In all of my ulcer cases a careful examination is made as to whether intestinal toxemia also exists. This is particularly true in the cases which undergo medical treatment for the ulcer. It holds true also for those cases which have been treated surgically, because unless one can clear up the intestinal toxemia there is danger of a return of symptoms in the upper abdomen. Such cases are relatively more numerous as surgery in the upper abdomen for ulcer is becoming more common. The wisest rule after the operation for the ulcer has been performed is to suggest a form of diet and to keep the patients under observation to see that they conduct themselves in a normal way. In addition, after the surgical treatment and a sufficient length of time given for surgical recovery, if an intestinal toxemia also exists that should be corrected as well. This is something I have learned within the last years and I am satisfied that it has tremendously increased beneficial results I have obtained in both medical and surgical treatment for gastro-duodenal ulcer.

Gall-Bladder Pathology.—In the instances of cholelithiasis the proper treatment is surgery, providing the symptoms can be ascribed to the presence of stones in the gall-bladder. These cases, however, should be recognized as having intestinal toxemia and even if the gall-bladder is entirely removed, the patient should be treated for the intestinal toxemia after the surgery is over. In instances of chronic cholecystitis on the other hand the treatment may be conservative from a surgical standpoint, the case considered medical and treated on the basis of the intestinal toxemia and continued for some time to see what result is accomplished in the case. If, then, the laboratory symptoms of intestinal toxemia clear up and the subjective symptoms of the individual continue, or get better and afterward return without a resumption of the intestinal toxemia, the case should be considered as requiring a surgical operation.

TREATMENT OF INTESTINAL TOXEMIA.

The alimentary canal with its predigested food material and warm body temperature, darkness and moisture, and doubtless anaërobic condition is an ideal culture bed and incubator for bacteria, and as a result, the bacterial flora of the alimentary canal of an adult is a particularly rich one. Kendall has shown that in an instance where there is a monotonous diet in which the protein is in the minority and the carbohydrate predominates, the bacterial flora is quite homogeneous. In the adult, however, where protein is quite apt to predominate in the diet, the bacterial flora is found to be heterogeneous. This is due to the fact that in case of the carbohydrates the splitting products are few and simple, whereas those from protein are complex, numerous and varied in composition. It is a well-known fact that the bacteriology of the human intestinal canal is quite facultative, and therefore in a putrefactive condition a carbohydrate and fat diet may be given which in the course of time turns the character from a putrefactive to a fermentative state of affairs; due to the encouragement that the change in diet brings about in the growth of such bacteria, perhaps of quite opposite groups. Then, too, organisms can actually change their metabolism and accommodate themselves to a change of diet. It is not uncommon to see organisms accommodate themselves to protein and then to a complete carbohydrate regimen. These changes consist essentially of alteration between proteolytic and gas-forming bacteria on a protein diet and acid-forming bacteria on a carbohydrate regimen. Often for a time, however, the absence of carbohydrate prevents the development of acid-forming bacteria on a protein diet, and the excessive amounts of acid by the fermentation of sugar prohibit the growth of the proteolytic aërogenic forms in the carbohydrate regimen.

To show how toxins of certain pathogenic bacteria, diphtheria, coli, and gas bacilli are altered by their facultative metabolism, Smith showed that diphtheria bacilli attack the carbohydrate preferably to protein when both are present in the same culture media. When carbohydrate is present in sufficient amount, the bacilli produce an acid which may prevent further growth and comparatively little toxin is produced, but if only a small amount of sugar is present, the bacteria get a good start, do not form acid enough to do themselves harm, and then they attack the protein and there is a marked production of toxins. This law is generally true with all of the bacteria. When the colon bacilli are grown in the sugar-free protein broth there results a disagreeable odor, a strong alkaline reaction, indol, skatol, aromatic

acid and other products of protein putrefaction, and these products, as is well known, are toxic when absorbed, and when present in considerable quantity, especially when there is an injured mucosa, they are readily absorbed. On the other hand, when the colon bacilli are grown in sugar broth the odor becomes agreeable, the reaction acid, and, in addition to carbon dioxide and water, lactic and small amounts of succinic and acetic acid are to be found.

From these facts it is readily seen that the character of the foods taken in the alimentary canal will very profoundly alter the bacterial flora and the toxins of the canal. It might also be added that there are many cases in which, whatever the diet, whether protein or carbohydrate, the bacteriology continues in spite of the character of the food, and it should also be remembered that bacteria may become facultative and may be quite as injurious in a fermentative sense as in a putrefactive.

Hull and Rettger have shown that milk undoubtedly owes its beneficial action to the lactose which constitutes nearly one-half the solid matter. When lactose is taken in liberal amounts by a human adult along with the daily foods the flora characteristic of healthy persons on a mixed diet begins to change until B. acidophilus appears in conspicuous or even dominant numbers. B. bifidus is always less common. Without the ingestion of lactose no such changes are observed. Following the use of laxatives these organisms are present in much larger numbers. This is ascribed by Hull and Rettger to the transfer of lactose along the alimentary canal so that before absorption a sufficient amount reaches the lower part of the intestine where the aciduric bacteria are known to multiply most abundantly under the right condition of nutriment and environment. Obviously, unless the sugar can be transported to a place where the most marked bacterial changes, putrefactive and otherwise, occur, no benefit will be brought about. This is important in connection with stasis, because manifestly when there is a stasis the giving of lactose would not be beneficial in stimulating bacterial acidification.

Intestinal Antisepsis.—For a good many years the medical profession has attempted by means of medication to accomplish an ideal which is not possible considering the many factors which are found present in the intestinal tract. The germicidal effect in the intestinal canal is an ostrich proposition. The substances used for this erroneous purpose have been many. Myriads of drugs and methods have been advanced, none of which have stood the test of time. It must be manifest that in a canal of intestines over twenty feet long in which a number of phenomena take place such an idealism as accomplishing

intestinal antisepsis by any single method is entirely out of the question.

Probably Bouchard,[32] more than any other, is to be regarded as the pioneer in intestinal antisepsis. Attempts were made by him to lessen putrefaction by administrating charcoal, naphthalin and iodoform internally. Baumann,[33] who used the method for determining the ethereal sulphates in the urine as the gauge of absorption of putrefactive substances, was probably more responsible than any one else for putting calomel so high in the list of the so-called intestinal antiseptics, a place it has no right to hold based on any distinct antiseptic action.

A great array of drugs has been tested culturally, and reports *pro* and *con* have been expressed by the authors. For instance, Sucksdorff[34] claimed to have obtained a drop of one-third in numbers of colonies in his own stool after taking 3.6 grams of quinine in divided doses; and even a more marked drop followed the administration of 2.1 grams naphthalin. Kumawaga,[35] using 14 grams acetanilid, U. S. P., on a dog, reported a reduction of colonies to the proportion of thirty-seven to one. Salkowski[36] got a considerable reduction of bacteria in the feces of a dog fed at intervals with chloroform water. Stern[37] found no effect following naphthalin, betanaphthol, salol and camphor. Von Mieczkowski[38], in cases of ileocecal fistula, tested the antiseptic values of bismuth, silver citrate, tannopin, betanaphthol and menthol by mouth. With the exception of menthol, his results were of no value; in the case of the latter there appeared to be a substantial reduction following the giving of such amounts as from 10 to 13 grams in divided doses. As early as 1911, Assmann[39] reported negatively on guaiacol cinnamate (styrakol), tannin-formaldehyde (tannoform), tribromphenol-bismuth (xeroform) and bismuth subgallate, using the culture method to gauge results. He employed dogs fed on milk-rice and meat-rice diets. In the same manner he tried the effects of two different silver-protein solutions (so-called "colloidal" silver solutions), which had been superficially tested by Klimmer[40] in 1900. If given in a solution of gum acacia along with the food, the results proved encouraging as the colonies per milligram of feces fell at times quite low. In the giving of colloidal silver preparations to man, it ought to be borne in mind that absorbed silver might be ultimately in part laid down in the skin. In the dog, Klimmer states that absorbed silver is laid down in the intestines, liver and spleen, but not in the nervous system, muscles, ligaments or skin; in the dog the skin is devoid of sweat-glands, and this may be sufficient explanation why no silver is deposited there.

By the Strasberger[41] technique the following agents have been literally "weighed in the balance and found wanting;" naphthalin, thymol, silver citrate, beta-naphthol and salicylic acid (doubtful), by Strasberger; magnesium dioxide and iodo-anisol by Feigen[42]; in cases of intestinal catarrh, negative results were obtained by Friedenwald and Leitz[43] using salicylate of bismuth, salol, potassium guaiacol-sulphonate (thiocol), acetyl-salicylic acid (asperin) and thymol; while favorable results with bichloride of mercury and ichthyol albuminate (ichthalbin) were noted; but the sharpest fall in bacterial excretion was obtained in a case of intestinal catarrh with hyperacidity when the patient was simply put on a change of diet. Berger and Tsuchiya[44] report a lessened bacterial output by the use of a patented preparation of agar-agar and hydrogen dioxide. Steele[45] found that in normal persons the administration of beta-naphthol and bismuth salicylate caused a slight fall in the bacteria excreted.

Manifestly, it is most unwise to expect changing the status of affairs in the intestinal canal by means of any medicinal substances by mouth. This entire method and line of endeavor should be given up as unscientific and impractical. Such benefits as are brought about by use of the so-called intestinal antiseptics, or in combination, have been accomplished entirely along the lines of effect upon the patient's mind, and not due to biochemical change that these drugs bring about. One deals with so many factors; the types of food, their physical as well as chemical characteristics, the age of the individual, the condition of the general body at the time, the physical expenditures in the muscular system in as these correct small digestive errors for a balancing nutrition of the tissues, the various phenomena of digestion, the transit through the gastro-intestinal tube, the chemical changes that are brought about by the various secretions that are poured into the canal, the incorporation of the bacteria with food, perhaps mucus and the segmentation of the food in various sections of the canal, which a single dose or even small corporated doses would not affect, that to continue the giving of intestinal antiseptics should be discouraged.

There is a great difference in the proportion of a disinfecting agent required to destroy micro-organisms and that needed to restrain development. For example, the germicidal strength of creosote is about 1 to 300, but it is distinctly inimical to bacterial growth when present in the proportions of one part to 4000. The following table gives the generally accepted strengths in which some of the intestinal antiseptics are positively efficient, and the dose this would represent for a volume of 6000 cubic centimeters:

	Antiseptic strength.	Dose required.
Beta-naphthol	1 to 10,000	9 grains
Copper sulphate	1 to 1100	80 grains
Chlorine water (U. S. P.)	1 to 16	12 fluidounces
Creosote	1 to 3000	30 minims
Phenol	1 to 700	3 drams
Salicylic acid	1 to 1000	90 grains
Phenyl salicylate	1 to 800	115 grains*
Solution of formaldehyde (U. S. P.)	1 to 2800	31 minims
Resorcinol	1 to 2000	45 grains
Thymol	1 to 1500	60 grains

* This figure is based on calculations from the amounts of phenol and salicylic acid in this compound. Bouchard found by actual experiment that it required 75 grains.

These figures are based on the supposition that all of the drug administered will remain as such in the intestinal tract, which is, of course, contrary to the fact. The questions of the rapidity of chemical change and absorption become of much importance in judging the efficacy of this group of remedies. Is there evidence that any of the so-called intestinal antiseptics can remain in the alimentary canal long enough to exercise their antibacterial influence? There is abundant proof that many of these substances are absorbed, in part at least, in a comparatively short time, but the evidence that any of them tarry for a considerable period in the bowel is far from satisfactory. The most important work on this subject that I know of is that which is offered by the ingenious experiments of Mieczkowski.[46] From the presumptive evidence from this it would seem that the conclusion is justified that theoretically it is possible to exert a mild degree of antisepsis in the intestinal tract, although it is unreasonable to expect any distinct germicidal influence.

In the experiments which Sucksdorff[47] conducted, an effort was made to influence the number of bacteria in the alimentary canal by plating a weighed sample of the stool and counting the number of colonies in the usual manner of estimating bacteria and administering one of the following in the table that he gives:

Weight of Bacteria.

Case.	Without drug.	With drug.	Drug.
1	7.44 grams	3.26 grams	Bismuth salicylate
2	5.0 grams	1.15 grams	Bismuth salicylate
3	2.74 grams	1.17 grams	Bismuth salicylate
4	2.23 grams	0.90 gram	Beta-naphthol
5	2.51 grams	1.44 grams	Beta-naphthol
6	1.69 grams	3.51 grams	Beta-naphthol

From the above, three substances turned out as being efficient in doses that are within the limits of safety. These are beta-naphthol, formaldehyde and creosote. It is probable, however, that formaldehyde is taken up with great rapidity, so that a practical intestinal antiseptic action would not be useful. Creosote, on the contrary, is absorbed with a fair degree of rapidity. It is possible by enteric coating to delay the absorption from the intestinal canal. Beta-naphthol is rather an insoluble substance, probably somewhat more soluble in the content of the bowel than in pure water. It is evident, however that a substance which is so sparingly soluble as this, must go into solution in the intestines very slowly, and therefore linger in the bowel for a considerable length of time. For this reason, as well as for the fact that even in very dilute solution it exercises an antiseptic influence, it would seem to be the remedy of choice in cases in which we wish to influence bacteria in any part of the intestinal tract below the upper duodenum. Sternberg[48] found that a 1-to-16,000 solution prevented the germination of the cholera spirillum, which a 1-to-24,000 solution failed to do. These concentrations would correspond to doses of about 9 grains, 5 grains, and 3 grains, respectively.

Harris,[49] giving the technique of bacteria counting before and after, reports the following: (See opposite page.)

General Medicinal.—Fatigue would have to be controlled by means of the well-known methods of rest, sufficient feeding, perhaps a sojourn in the country, tonics of various sorts, and those which are metabolically constructive as malt, the oils, hyperphosphates, etc. and the hematinic forms of tonic. Persons actively engaged in social ways, in home duties or in business should have their affairs curtailed. Regular living and regular hours with a large amount of rest at night whether sleeping or not, are in order.

The anemia is controlled by means of hematinic tonics of which the best forms are the inorganic forms of iron, hypodermically administered. Usually this is required but for a short time. A diet heavy in organic iron such as meats, or a heavy iron-bearing vegetable diet and fruits would answer the purpose in different instances.

Anorexia is controlled best by the use of elixir tinct, ferri chloride and gentian well diluted before meals. Other forms of hematinic tonics might be in order for the purpose. Of course, the patient should be encouraged to eat sufficient amounts of food.

Insomnia which is often a distressing factor in these conditions had best not be controlled by hypnotic substances, excepting perhaps for a few days at the beginning of treatment. The insomnia usually disappears more or less as the intestinal condition improves. Of

RELATIONSHIP OF BACTERIA TO STOOL, FOUND BY STRASBERGER TECHNIQUE (HARRIS' FINDINGS)

SALOL TEST

Patient.	Date.	Drug Dosage.	Feces.		Bacteria.		Colonies in Millions. Per Gram.	Indican In Urine.	Appearance of Carmin Indicator.
			Total Moist.	Total Dried.	Total Dried.	Per Cent. Relation.			
Rb.	3/4/12	0	130	9.3	0.7	7.54	167	+ + +	0
	3/8/12 } 3/9/12 }	6.0	390	101.79	4.13	4.06	53	+	0
	3/10/12	0	165	34.48	6.4	18.56	110	+	+ + +
Mt.	3/7/12	0	785	†	†		150	+ + +	0
	3/8/12 } 3/9/12 }	6.0	0 205	26.65	2.62	9.41	840	+ +	0
	3/11/12	0	650	131.49	3.64	2.76	820	+ +	+ + +

BETA-NAPHTHOL TEST

Ma.	3/25/12	0	140	28.89	1.31	4.55	30	—	0
	3/26/12 } 3/27/12 }	4.0	80 185	† 42.60	† 2.14	5.02	120 34	— —	0 0
	3/28/12	0	40	4.40	0.28	6.54	460	—	+ + +
	3/29/12	0	175	31.90	1.65	5.19	214	—	+ + +
	3/30/12	0	180	28.88	3.09	11.1	87	—	0
Ro.	4/27/12 } 4/28/12 }	4.0	329	77.08	14.27	18.51	320	+	0
	4/29/12	0	185	12.32	2.84	23.11	700	+ + +	+ + +
	5/1/12	0	‡	‡					
	5/2/12	0	235	8.15	2.39	29.41	10	+	0
	5/3/12	0	155	14.38	2.17	15.09	37	+	0

GUAIACOL CARBONATE TEST

Zm.	4/27/12 } 4/28/12 }	4.0	35	8.66	1.92	23.38	950	+ + +	0
	4/29/12	0	90	14.99	4.14	27.67	355	+ + + +	+ + +
	4/29/12 *	0	150	12.46	3.18	25.44	85	0	+ + +
Kf.	5/6/12 } 5/7/12 }	4.0	‡	‡					
	5/9/12	0	345	84.73	11.86	14.00	500	+ + +	+ + +
	5/10/12	0	100	14.97	1.94	12.95	540	+	0
	5/11/12	0	160	38.24	3.77	9.84	670	+	0
	5/14/12	0	180	22.19	5.07	22.87	30	+	0

* Further observations impossible, as patient was transferred to surgical ward. † No determination. ‡ Stool lost. — No test.

some value is the well-known hot spinal douche before retiring, it usually taking about fifteen minutes until complete relaxation of the nervous system has taken place. Occasionally bromides are in order and perhaps a mixture of bromide and valerian through the day may encourage better sleeping at night. Various skin conditions would require the necessary lotions and ointments. Marks of neurasthenia require special attention, perhaps a rest in bed treatment, although those who are up and about and not distinctly exhausted, would be benefited by the use of the various tonics hypodermically given. A resulting gastric hyperacidity or hypersecretion might require the use of alkalies. An atony may be so distinct that large doses of nux vomica, strychnine, together with the use of the sinusoidal current, would be necessary adjuncts to the treatment. Perhaps a gastric hyperesthesia, or one generally in the nervous system, would require bromide and valerian to control the distress.

Hydrotherapeutic and mechanical measures such as baths, electricity, massage, etc., may be employed. Such procedures are required to build up the general tone of the body, and where the means are not at hand, it may be necessary to send the individual to an institution equipped with apparatus for the purpose. In such instances there is often a complete mental diversion from business and family cares required, and those patients who have been depressed, become interested in golf, touring, music, reading, etc., which bringing a more cheerful atmosphere, has a beneficial effect upon them. Very important, however, in their daily routine is exercise. In the saccharo-butyric cases the individuals are often quite obese; exercise and dieting for the obesity would be in order. Such exercising should be of the heavy type so as to put as much strain on the muscular system as possible. In the indolic and mixed forms there is usually so much fatigue, devitality, etc. that strenuous exercising is contra-indicated. In such instances, however, some exercise should be carried out and the system that I would recommend is a combination of massage and exercise at the same time, the various motions being as follows:

1. Rub each foot on top with the other, at the same time rubbing the neck with the hand.

2. Stroke each arm alternately from the shoulder on the upper side down to finger tips, continuing underneath up again to armpit, then down same side of chest, and give a short stroke behind shoulder under armpit.

3. Without bending the knees, bend the trunk forward and stroke from ankle up front part of legs, stomach and chest to the neck, at the

same time raising the trunk; then stroke down chest to diaphragm; now bend trunk forward and grab around the back, with hands on each side of spine, as far as possible, and stroke from there down over lower back, continuing down back part of legs to heels.

4. Stroke with both hands from each side of knee alternately up over side of hip and loin, then straight across the abdomen with the one hand, and the diaphragm with the other.

5. Press the arms and hands alternately with a swinging movement from behind down on something in front and on a level with the chest, at the same time giving the trunk a quarter turn to the side, and rubbing the kidneys and lower back with the back of the other hand. (If swinging and pressing with the right arm and hand turn to the left and rub with the left hand, and *vice versa*.)

6. This is similar to No. 5, but here swing the one arm sideways and press sideways on something in front of you, at the same time rubbing the left and right side alternately with the other hand. When rubbing the left side with the left hand from hip up over diaphragm, the trunk is turned to the left, and the sideway swinging and pressing is done with the right arm and hand, and *vice versa*.

7. Lift the knees alternately up to chest, then while legs go down stroke both sides of legs from ankle up over abdomen and chest to neck; then go down the spine with the back of one hand.

8. Bend the trunk to right and left, at the same time stroke both sides from side of hip up to armpit with each hand alternately (heels together).

9. Jerk the trunk to right and left, at the same time rubbing both hands across the chest.

10. A rolling of the body at the hips, the hands making pressure on the abdomen on the relaxed side.

Practice a breathing exercise between each massage exercise except in No. 8, when legs remain stationary and feet are kept at least a foot apart and nearly parallel. Do not exercise for at least one hour after a meal.

As a special breathing exercise: Inhale as deeply as possible with hands placed on loins and elbows, and shoulders thrown back, at the same time rising up on toes and bending the knees so that the heels touch the seat. Exhale the air through the mouth while rising up on legs and toes and down again on heels.

Intestinal Irrigation.—Intestinal disinfection has been tried by means of the trans-intestinal lavage method using various solutions for the purpose. What is accomplished here is a duodenal lavage and the benefits obtained are undoubtedly due to the washing of the

intestinal content onward, and not to the various solutions which are used. Jutte uses a combination of saline cathartic with phenolphthalein. Other men claim benefit from the use of a solution of magnesium sulphate. There is no doubt that these methods accomplish a thorough cleansing of the lower end of the small intestine and all of the large, and it is my belief that such results as are accomplished have been brought about by the mechanical use of the water plus a purgative effect from the salts in solution, and not from any bactericidal action. In this limited sense they are worth the while, particularly in the saccharo-butyric cases where there is a high anaërobic content, a large amount of Welch bacilli and perhaps Gram-positive diplococci or single coccal growths. In my experience the use of the trans-intestinal lavage method of washing the small and large intestine is also worth

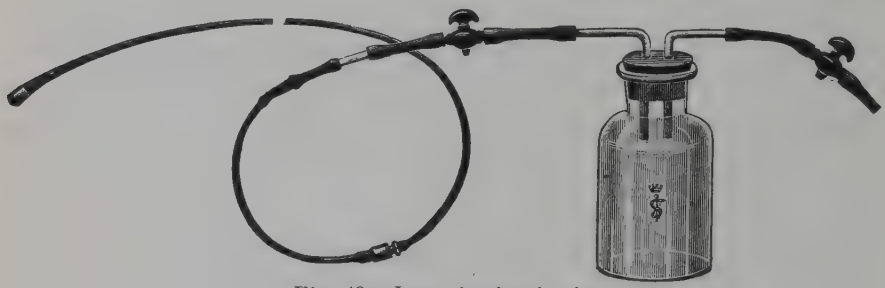


Fig. 48.—Jutte duodenal tube.

while in those instances of saprophytic infection, both of the Gram-negative and Gram-positive types. There is no doubt that when its use is persisted in for a length of time these individuals are very much relieved of the symptoms for which they come under observation. But as a general method of treating intestinal toxemia I doubt that it could serve any purpose which would be curative or helpful in a large enough number to warrant its general use in all cases. The Jutte tube and method for this is by far the best.

Colonic Irrigation.—What has been said in connection with trans-intestinal lavage should manifestly hold more true in colonic irrigation. Numbers of men that I know of are engaged in irrigating the colon in instances of colitis, intestinal toxemia, etc., and I have yet to see the case after an extensive study of this method that has been benefited by it. There are not a few individuals who believe that they have been benefited, the effect being entirely one of purgation and mental suggestion and not due to actual curative benefit brought about by the method. Of course an emptying of the intestinal canal has been accomplished but this can be done quite as well by any patient

using an enema, simply lying on the left side or perhaps in the knee-chest position. No physician, nurse, high rectal tube, or any definite solution employed is worthy of consideration in these cases as a method of treating intestinal toxemia while under a physician's attention. It does not pay for the time and effort the medical man puts in on the effort, nor the patient for such as they have to pay for the treatment.

In my work I do not use any form of rectal irrigation, either accumulative or by recurrent tube. It is my belief that the human intestinal canal is not made for the purpose of withstanding large quantities of water. Many times distinct harm is done, and such results as may be brought about by a benefit in the pathology of the gut are so few that they are not worth the while, and could quite as well—in fact, much better—be accomplished by means of an anti-constipation diet, with perhaps the addition of slight purgative assistance which would insure the bowels moving normally.

The use of vegetable purgatives, particularly the drastic vegetable purgatives, should be entirely discouraged in cases of intestinal toxemia. It is my observation that they do more harm than good, and while patients may feel relieved after their bowels have moved, in the end they are more toxic than they were before beginning with the drugs. An occasional small tap-water enema may be in order every week or so, but its regular use never.

Intestinal Putrefaction and Water Drinking.—Data is on hand which indicates a marked decrease in the output of bacteria in the feces when normal persons were caused to increase their water ingestion to 3450 cubic centimeters per day, the water being taken with meals. That intestinal putrefaction was also diminished under these conditions is indicated by an accompanying decrease in the urinary indican values during the interval of high water intake. The course of the total ethereal sulphate excretion did not parallel that of indican, thus furnishing evidence in favor of the view that indican has an origin different from that of the other ethereal sulphates. When a normal man passed into a seven day fast from a high protein level, it was found that the daily output of fecal bacteria was markedly lowered, with a return to normal values with the inception of a post-fasting period of low protein character. Indican and total ethereal sulphates are also decreased under the fasting regimen, this decrease being followed by an increase on subsequent ingestion of food. (Blatherwick, Sherwin and Hawk in Journal of Biological Chemistry). That the use of water is worth while is evident but in cases where there is ptosis or in which there are marked states of atony the use of

large amounts of water may be contraindicated. Water-drinking, however, cannot cure an intestinal toxemia, or even materially benefit it if it is distinctive.

Dietetic Treatment.—In the three types of toxemia one must avoid continued reinfection that follows the ingestion of putrefactive bacteria with the food, promote prompt digestion and rapid absorption from the small intestine, and reduce the number of putrefactive anaërobes in the ileum and colon. To avoid the infection and reinfection, the mouth must receive scrupulous care. Carious teeth and gingivitis must be treated carefully by the intelligent use of tooth brush and of washes containing peroxide of hydrogen, camphophenique, or a weak solution of camphenol. Gastric lavage may be necessary in addition, perhaps best conducted in the morning. Oral sepsis requires strict attention.

The preparation of food and ordinary cleanliness is very effective —it is probably better to use cooked food as much as possible. Fruit is not above suspicion, for on the surface of most raw fruits bacteria swarm. The bacillus of malignant edema, for instance, being commonly present on the banana peel, and the B. putrificus on grape skins. Raw milk always contains a large number of bacteria and often some of the putrefactive forms, especially B. putrificus. Sterilization of milk is of little value. Pasteurization or the ordinary boiling kills the lactic acid formers but does not harm the spores of the putrefactive organisms. Cheese contains many putrefactive forms and is best avoided, particularly inasmuch as many of these patients lack the protective action of a normal amount of hydrochloric acid in the stomach.

With rapid digestion and prompt absorption little pabulum for the putrefactive organisms reaches the colon. These processes are often facilitated by means of the secretory and motor functions of the stomach. Chief in importance here is proper mastication which largely determines the ability of the body to utilize food. When large masses of meat are swallowed they commonly appear in the feces. The comminution of food outside of the body is not an adequate substitute, for the patient then loses the emotional stimulus to gastric secretion, and also the digestive action of the saliva as well. The administration of hydrochloric acid helps for a time but in long standing cases, especially those of the combined indolic and saccharo-butyric types, it is of little use. Ferments such as pepsin and pancreatin are of doubtful value. Diastase which enables a patient to utilize more carbohydrate might be worth the while. If the stomach is hypersensitive, or a status of irritability exists, it is advisable to give more

meals and to administer flaxseed or some other demulcent before eating.

It is a good rule to follow that putrefaction in the intestines is directly proportional to the amount of proteins in the food. This is obtained from meats and from vegetables. The vegetables are comparatively safe, however, this being due to the fact that vegetable proteins are not so accessible either to the alimentary or bacterial enzyme, and therefore are not so readily decomposed, and also to the fact that bacteria utilizes the carbohydrate substances in preference to protein. There is some difference in the effect of carbohydrate foods. Bread, sugar, potatoes, and legumes often give rise to most of the organic acids and gases, whereas on the other hand, rice, sago, tapioca and arrowroot give rise to comparatively little fermentation. As a rule cheese should be forbidden as well as most kinds of fish, game, pork, veal, hashed meats, and made-up dishes (particularly those that have stood over night) stews, rich gravies, soups, meat extracts, pastry, cake, tea and coffee.

So much has been written upon the subject of diet in connection with intestinal toxemia, and so little is worth while, that I beg to be excused quoting from the literature of the past, and will undertake to present only my own views. In a word, there is no definite method of dieting for intestinal toxemia, and it may also be added that there is no definitely indicated diet which would be helpful in all instances of the same type of condition. The best rule is to plan a diet according to the type of intestinal bacteriology that is present, the designation being according to whether the toxemia is putrefactive, fermentative, or of the mixed form, and then not to depend upon the diet alone or continuously. The rule I follow is to plan a normal diet, keeping the quantity of total protein down to not more than 60 grams in a day, roughing up the diet so as to overcome the element of constipation and the presence of a colitis if it exists, adding calories in the shape of fats when there is distinct debility or loss of tissue, the use of a high protein diet in saccharo-butyric infections, and the employment of a diet which gives the minimum amount of food, allowing a general selection in cases of the mixed types. By following this plan I have been fairly successful in dieting instances of intestinal toxemia, not having the bother of making out an individual diet for each patient, or selecting various foods for this or that case. After making out individual lists along general lines for years I have come to the conclusion that I have largely hoodwinked myself. I now go upon the findings in the laboratory and X-ray examinations and make up a diet according to the type of toxemia, the abdominal and general re-

quirements of the individual, always encouraging the taking of sufficient amounts of food.

The type of diet I use in putrefactive cases where putrefaction is marked is somewhat on the order of the following:

The plan of the diet is to partake only of fluid or semi-solid foods, at three or four hour intervals during the day and evening, and preserve a regularity in the taking of them. The vegetables (no rough forms), starches and cereals must be well boiled, and mashed on the plate. The quantity taken at a time should be moderate, and they should be taken slowly. The meals should be begun at 8 A.M. and finished at 10 P.M. Among the foods best to take are the following:

ALBUMINOUS DRINKS.

Egg broth	Grape juice	Mutton broth
Egg nog	Grape juice and egg	Nutritious beef broth
Junket egg nog	Malted milk and egg	Broth with grains
Albuminized water	Kumyss	Egg broth
Albuminized clam water	Zoolak	Cocoa
Junket	Rice milk	Malted milk cocoa

SOUPS.

With or without noodles, crackers or croutons.

Cream of celery soup	Mock bisque soup
Celery soup (gum gluten)	Green pea soup
Asparagus soup	Rice soup
Corn soup	Victoria soup (with broth)
Tomato soup (with broth)	

CEREALS.

Flour gruel	Rice, farina and oatmeal gruel
Porridge	Gum gluten breakfast food
Cracker gruel	Corn meal mush
Barley gruel	Hominy mush
Barley gruel with broth	Rolled oats
Arrowroot gruel	Steamed rice
Indian meal gruel	Boiled rice, farina, tapioca, sago

FRUITS.

Pineapple, baked banana, steamed rhubarb, baked apples and apple sauce, stewed prunes.

PASTRY.

Rolls, any kind; bread, any kind; cake or crackers, any simple kinds.

SHELL FISH.

Raw oysters, with lemon only	Clam bouillon bisque
Pan roast oysters	Oyster stew and soup
Creamed oysters	Broiled oysters
Scalloped oysters	

Eggs, four a day.

Soft boiled	Plain omelet
Steamed or baked	Foamy omelet
Golden rod eggs	Bread omelet
Egg nests	Poached eggs plain

Fish.

Creamed fish, baked or boiled fish (plain sauce).

Vegetables.

Boiled potatoes	Peas
Riced potatoes	Beans in purée form
Mashed potatoes	Lentils
Creamed potatoes	Spinach
Baked potatoes	

Desserts.

Soft custard	Gelatin
Meringue or floating island	Souffles
Banana, peach, or apple custard	Junkets, custard, cocoa, coffee, plain
Chocolate	Cornstarch pudding
Malted milk or baked caramel custard	Cornstarch, fruit jelly
	Chocolate or cocoa blanc mange
Gum gluten pudding	Plain or tapioca cream
Rice pudding, peaches and rice	Pineapple cream
Steamed and boiled rice	Pineapple, apple or raspberry tapioca
Rice meringue	Jellies (fruit and cereal)
Cream of rice pudding	Fruit whips
Bread and cracker puddings	

In most instances, however, more or less use of protein is allowed for many of these individuals require protein, and can take it providing it is in such form that it is readily digested and quickly absorbed so as not to accumulate in the colon. A copy of such diet, with constipation additions, together with the necessary increase in fats to encourage an increase in weight and strength is the following:

General Rules.—Care should be taken that all of the foods are fresh, cleanly cooked and served, and that no foods that have been standing some hours in a cooked condition are partaken of. The mouth should be cleansed with plain water (preferably with a little bicarbonate of soda dissolved in it) before and after the meals and when possible at other times. A thorough cleansing of the teeth and a correction of such dental conditions as may exist and the use of dental floss is advisable. Adopt the plan of taking either four meals a day, moderate in amounts, or three meals a day with supplemental meals between them and before retiring. Thorough cooking, cutting foods finely on the plate or mashing them, complete mastication and slow eating are advised. Foods should not be eaten under conditions of fatigue, mental excitement or depression and a rest for an hour after each meal is desirable. No condiments such as sauces, mustard, pepper, lemon, and so forth are allowed, and all foods should be plainly cooked and never in made

up dishes. The use of salt is allowed. When there is distress in the stomach drink a glass of warm flaxseed water before meals, and no fluids, including soup, milk, water and so forth are allowed with the meals, although these may be taken in the meal intervals; drinking of a glass of cool water about an hour after the meal is sufficient.

Foods Allowed: Bouillon, broth, consommes, purées. Any of the well cooked cereals served with milk sugar and fresh cream. Eggs in any form but not more than two a day. Breads, rolls, zweiback, biscuits and crackers. All foods made of gelatin, not more than two ounces of meat, poultry, game and fish in a day. Take a half pint of fresh cream, as much unsalted butter and olive oil as possible. Any of the vegetables may be taken, except potatoes, tomatoes, asparagus and canned vegetables, but they must be cooked to softness; peas, beans, and lentils being the most wholesome. The green vegetables and salads are allowed. Other foods of value are: custards, egg and milk, peeled fruits, jellies, marmalades, apples, pears, green chicory and spinach. Eat at least three of the following bran gems, well buttered, during the course of the day.

Bran Gems: One-half teaspoonful soda, saleratus, dissolved in ½ cup hot water. Add, when dissolved, ¾ cup molasses, then a tablespoonful of butter, salt to taste, 2 cups wheat bran, 1 cup bran meal, 1 cup milk, mix all the above ingredients together. Put in a muffin pan and bake 45 minutes in a slow oven.

Two added measures of moving the bowels are to take a dish of stewed prunes sweetened with milk sugar instead of cane sugar, or from a teaspoonful to a tablespoonful of white vaseline before retiring. If then the bowels do not move, inject about a half a tumblerful of olive oil into the rectum at night.

It may be found that apple sauce, sweetened with milk sugar instead of cane sugar, may be more efficacious than the prunes. If such is the experience, apple sauce may be used instead or they may be taken alternately, eating one on one day and the other the next.

Where distinct fermentation exists I use a high protein diet. A number of years ago the use of the beefsteak and water diet for the correction of intestinal toxemia had quite a vogue, and there is no doubt that considerable benefit was brought about by it. Such cases as improved were undoubtedly those of the saccharo-butyric fermentation and not the indolic or mixed forms, which manifestly would not be improved or even would be made worse by a diet high in proteins. A practical diet high in proteins is the following:

This diet is a temporary one. Take mostly meats—all forms of beef with the exception of cuts from the shoulder, kidneys and liver. The same is true of lamb. These meats should be fresh and taken in a broiled or roasted state. Mutton is permissible but no pork nor veal. May take any kind of fish broiled or boiled with the exception of shad roe and shell fish. May eat eggs in any form. Butter and whole milk are allowed, together with any form of simple cheese of the cream variety, such as Philadelphia, Neufchatel and cream cheese. Eat as much gelatin foods as possible. Oatmeal and rolled oats are allowed. May have breads or crackers made of gluten or rye flour. Lentils and dried peas are permissible. There is no objection to an occasional orange, pineapple or strawberries. The best drink would be chocolate and cocoa.

As was stated before, too much dependence should not be placed upon diet in the treatment of intestinal toxemia. Some benefit can be brought about of course, but the cure of the condition on the basis of altering the bacteriology is quite temporary, idealistic, and not steadily useful. Some benefit can be brought about in this way, but it is only transitory because ofttimes when a definite absence, low or full protein diet is indicated, after a course of time it will be noted that the bacteriology present in the individual has become facultative and has now changed the type of the toxemia from one to another. Those cases always suggest that an infection is present in the gut contents and mucous membrane of the small and large intestine and perhaps no dieting will be of use, or again that a definite pathology is present and therefore no diet will avail, or that there may be some error in the secretions and the individual is toxic more from the mucous membrane than from the content of the gut or from an infection in the mucous membrane. However, the plan as mentioned above, the absence of protein diet, that in which the protein is low, and the one in which the protein is high, and these based upon the study of the case in the nature of the process in a biochemical way is the most advisable that I know of.

Bacterial Treatment.—Beginning with a description of the vaccine work which is an important topic of the treatment, I desire to consider for a short time the use of the Bulgarian bacillus and other forms of organisms taken by mouth for the purpose of favorably influencing intestinal toxemia. While it is possible to duplicate the experiments of Metchnikoff in the test tube, it nevertheless is not possible to duplicate these test-tube experiments in the human economy. The use of the so-called B. B. culture for the treatment of intestinal toxemia is perhaps the greatest fallacy in medicine, and it is surprising to see how it persists from year to year, practically since 1910. There is no doubt that when there is a high anaërobic content in the intestinal canal, essentially a status of fermentation, that the use of B. B. culture is of some value. Numerous discouraging results from its use has brought more or less explanation in the way that the cultures used were not active or that they were not in the proper media, a controversy between the proprietary houses, one depreciating the worth of B. B. culture as put out by another house, and so on. The truth is that the use of B. B. culture for the purpose of favorably influencing bacteriology in the intestine is a myth, and is one of the most unscientific, wasteful procedures that I know of in medicine. Some slight degree of benefit can be accomplished by the use of the lactic acid bacilli, particularly instilled into the colon, where there is

a putrefaction in that portion of the intestinal canal. But even here the results are not worth the effort. What B. B. culture or the lactic acid bacillus culture instilled into the colon could accomplish, could be done more definitely by lactose sugar alone or by the so-called B. acidophilus. This organism which is recoverable from the stools of nurslings, particularly those that are bottle-fed, may be worth while, used in doses of three to five million taken after meals. After a rather steady use of all of these methods of administering bacteria for the purpose of influencing the bacteriology of the intestinal canal, I have given up their employment. They are expensive to the patient and accomplish nothing more than the plain lactose sugar could do very much better. It will be remembered that lactose encourages the growth of the aciduric bacteria in the small intestine.

Vaccine.—It is necessary here to consider whether the infection is simply of the intestinal content—namely, a true intestinal toxemia, or whether in addition to that there is an infection of the mucosa—namely, a distinct infection. When local pathology exists an infection of the mucosa is always present. It is probable that in the toxemia due to infection of the content wherein the mucous membrane and submucous tissues are in a resisting state that very few if any general or constitutional symptoms are present. The treatment of these cases is essentially along general lines, together with proper dieting, the use of intestinal treatments, and so forth. But where distinct infection exists, the vaccine theory offers a means well worthy of employment. When infection of the tissues has taken place, distinct constitutional symptoms are present. My belief is then that whatever may be done in a simple way unless vaccines are resorted to, but little if any benefit can be accomplished. Of course the dietetic means of controlling the infection within the content of the gut are in order however much pathology there may be present.

Immunity may be defined as non-susceptibility to a disease, or as the ability to resist the causes of the disease. The body may be immune because of inherited properties or because it has become so during life. In intestinal infection work the individual has lost his inherited properties of immunity and has acquired none during life. Therefore, both the natural and acquired immunity are gone, and one undertakes by the use of vaccine to give an acquired immunity, either active or passive. Active immunization is usually called vaccination and generally produces in the individual a modified form of the disease. The individual in this case produces his own immunity. In artificial or intentional inoculation the etiological factors, or more particularly the causal organisms injected, must be so modified

that the natural course of these will not follow the inoculation or injection. Acquired active immunity is produced by the injection of living or killed micro-organisms or of toxins produced by these organisms. It is not possible in vaccine intestinal work to produce passive acquired immunity. It also is not practical or worth while to employ the opsonic index method of estimating the resistance of the individual according to the work of Wright and Douglass.[50] There are so many forms of bacteria in the gut that the opsonic index results are generally not distinctive.

For centuries it has been known that following an attack of certain acute infectious diseases there remains a certain loss of susceptibility to the contraction of a second attack of the same disease. Early in the eighteenth century this experience was utilized in vaccination against small pox. This successful immunization can now be accomplished against cholera and typhoid fever, as well as conferring more or less benefit in other conditions.

Sir Almroth E. Wright, in his introductory address delivered before the Royal Society of Medicine on May 23, 1910 gave his conception of the rationale of vaccine therapy as the exploitation, in the interest of the infected tissue, of the unexercised immunizing capacities of the unaffected tissues. There exists a correlation between the vaccine and the antibacterial defenses of the body. This can easily be demonstrated by the opsonic index, which accurately measures the opsonic power of the blood, that is, the antibacterial defenses of the body (as stated above not advisable in practice in intestinal work).

The limitations of vaccine therapy, as Wright sees them, are: "1. Vaccine therapy can be applied only where an exact and complete bacteriological diagnosis has been made, and where the diagnosis is kept up to date. 2. It can be applied only by those who have some acquaintance with bacteriology, some understanding of the rationale of vaccine therapy, and a knowledge of the dose of the particular vaccine which it is proposed to employ. 3. A limit is placed to the efficacy of inoculation by the fact that there are definite limits to the responsive power of the patient. 4. Successful results can be obtained only where an efficient lymph stream can be conducted through the foci of infection. 5. In long standing infections vaccine therapy can give definite results only after a long succession of inoculations, and there is no security against a relapse until the infection has been completely extinguished."

Wright believes that a proper therapeutic dose of vaccine in an infected individual is followed by a period of intoxication, accompanied by a diminution in antibodies (negative phase), followed by a

stimulation of the immunizing machinery and the production of an increased amount of antibodies (positive phase). Theoretically, it should be possible to bring about the production of surges of antibody production, each wave higher than the preceding one, with progressive beneficial results to the infected individual. Wright, in his early work, insisted that, in order to provoke a positive response to vaccination it was necessary to inject a sufficient dose to produce at first intoxication (negative phase). He also believes that an interval of at least seventy-two hours should elapse between inoculation of vaccines because of this toxic-negative phase following each inoculation. My experience and that of others has shown that with proper dosage the ideal immunizing response can be produced without the toxic negative phase. Physiological doses of vaccines should be followed by an immediate production of antibodies without toxic action.

It has been generally accepted that the immune bodies are produced almost wholly by the blood-making (hemapoietic) organs, and thence delivered into the blood stream. This theory fails to take into account the special immunity which certain tissues exhibit against infection, which in the intestines is definite.

The most obvious objection to the use of vaccines in general infections is that the patient is undergoing extreme intoxication and that the injection of vaccines will but add to this intoxication. This is not so in intestinal work. Another objection offered to the use of vaccines in general infections is that vaccines stimulate the production of bacteriolytic substances and that these substances may kill many bacteria and set free their toxins, thus overwhelming the body with toxic products. I have never seen reported harmful results relative to the sudden setting free of their toxins. Following the injection of vaccines, the theoretical evolution of bacteriolytic substances, if occurring, must take place slowly and gradually.

A third objection is that patients undergoing infection are in an anaphylactic state, and therefore are hypersusceptible to intoxication. With chronic infections such as tuberculosis this danger apparently exists, and for that reason I believe the use of tuberculin in treatment should be carried out with extreme care. I have never seen evidence of its existence in intestinal conditions produced by other bacteria than T. B. It takes time for the establishment of anaphylaxis, and the general infections with their sudden onset should be largely free from such danger. Anaphylaxis is a purely theoretical danger, as is evidenced by the infrequency of serious results following the use of diphtheria antitoxin.

Christian Herter's work on the bacterial infections of the digestive tract appeared in 1907. Sometime between that and 1910 Allen and others wrote on vaccine therapy. Recognizing the possibility of being able to favorably affect the intestinal infections in man by means of vaccine methods of treatment, I engaged in the clinical application of autogenous bacteria according to the infection I believed existed in intestinal toxemia, carefully noting the results. All of the work done was by the use of autogenous colon vaccine, administered both subcutaneously and by way of the rectum. Chvostek[51] had given colon vaccines by mouth, and when some very remarkable results by injection of autogenous colon bacillus vaccine had been accomplished by me (N. Y. Medical Record, Sept. 24, 1910), Turck[52] suggested their use. In 1910 I had treated one hundred and twenty-seven cases of distinct intestinal toxemia by means of autogenous colon vaccine with a number of striking results, some indifferent results, and a few failures. I suggested that it might be possible to influence the cases better by use of autogenous colon vaccine administered by rectum and using the viable form, employing much larger doses than possible by subcutaneous injection, the initial dose of which is limited to between 25 million and 50 million, given every fourth day and gradually increased. The colon route was also deemed advisable because of some very severe reactions, local and general, in the subcutaneous injection method. It was observed by me that as many as from 5 trillion to 30 trillion viable colon bacteria could be given by rectum without much reaction, and that a leucocytosis was possible of accomplishment in the same way. Also that the resulting leucocytosis was more steady than that by subcutaneous injection, and if the vaccine could be kept up for three or four months the results were quite as good—in fact were much better than by the cutaneous route. Since that time Satterlee[53] has reported good results by the use of colon bacillus vaccine given subcutaneously.

The original use of the colon vaccine by me was suggested by the conclusions drawn by Herter that the colon bacillus was capable of initiating the toxic process in the intestine, although it usually did not finish the putrefaction, this being accomplished by other forms of anaërobic growths. It is a well-known fact that many strains of the B. coli are beneficial in the intestinal canal and inhibit putrefaction and fermentation rather than initiate or encourage it. The whole question is bound up in the strains of the coli, and it has been my experience that there are essentially 4 pathogenic types in the 17 different strains. It is a well-known fact, for instance, that the colon bacilli recovered from the human intestinal canal is the most virulent of any.

This statement of Deaver's[54] is true in the main, but depends upon the strain of coli one is describing. Unfortunately the control test in the diagnosis of colon bacillus infection, the serum agglutination test, would be valuable if it were reliable, but its reliability cannot be depended upon. One can always recover B. coli from the intestinal canal of man. The point is, in this subject to figure as to how much the coli has to do with initiating a process of toxemia, and whether on the other hand the coli present might not after all be innocent forms and distinctly inhibitory to other forms of pathogenic organisms. This can only be accomplished by laboratory procedure in isolating the various strains and studying them culturally as well as by injection in guinea-pigs and other animals.

Having concluded that the B. coli have to do with the production of symptoms in the case, and decided upon a subcutaneous administration, the process of treatment is simple. The patient is given a dose of castor oil and the third or fourth stool following is taken to make the vaccine from. As many colonies and strains as possible should be used to make the vaccine, which would be a polyvalent autogenous emulsion and perhaps the infecting strain given.

Depending upon the age and clinical conclusion as to the vitality of the individual, the initial dose is from about 15,000,000 to 25,000,000 of dead bacteria subcutaneously. The dose is repeated every four to seven days, and a gradual increase of about 20,000,000 each time until the maximum of 200,000,000 and 300,000,000 bacteria is reached. One does not see much, if any improvement until several doses have been given, and in fact it may be necessary to go on for two or three months before results are accomplished. There is a certain reaction after each dose, which consists of local redness and swelling, with perhaps a considerable spreading until a large area is involved. The local reaction begins on the same day and may last for two or three. In severe cases of intestinal toxemia considerable reaction may occur, and one would have to reduce the quantity of coli administered at a dose. The good results when brought about are probably accomplished by an immunization and sensitization of the body cells.

Because of the failure in many instances of the use of autogenous coli vaccine subcutaneously administered I believed that the rectal route would be more worth a trial, and so far as I know, I was the first one to use the subcutaneous and the rectal route for influencing conditions of intestinal toxemia by the administration of coli vaccine. I desire to quote from the article published on the rectal instillation method which holds almost as true today as it did in 1910, and after about 2200 cases have been treated.[55] "My interest in the use of a direct bac-

terial method of treatment was suggested by the uniformity of the different bacterial pictures seen in examining specimens of normal and abnormal stools stained by the Gram differential method; the fact that the coli bacilli grow only for a certain time in bouillon, when, probably because of their generation of thermostabile and thermolabile substances allied to phenol, their proliferation is inhibited and they become quiescent or resting but not killed (the latter was a confirmation of Conradi and Krupjurveit observations with the Bacillus coli communis and the Bacillus lactis aërogenes (the last of which organism probably suggested the use of the Bulgarian form as a germicidal bacteria against all others) and also, the fact that Coli bacilli were most numerous in stools of normal individuals, but were diminished or absent in some cases of excessive chronic intestinal putrefaction, having excess of indican in the urine, even when the intestinal contents have somewhat rapidly passed through the colon.

I have come to the following conclusions: In cases of chronic intestinal putrefaction wherein carcinoma, colonic obstruction, abnormal organic disease of the pancreas or stomach, or gastrointestinal atrophy, etc., are not responsible for the condition, much benefit can come from raising the content of B. coli communis in the gut by instillation either of the autogenous mixed forms or strains from other individuals; whether this is due to a real antagonism between the toxins of the B. coli and the other putrefactive organisms, these toxins being existant in the cultures injected (which bacteriologists claim is slight in amount with the B. coli), or whether the B. coli so injected are directly toxic to these other bacteria I am not prepared to say. (We know that the dead as well as the living B. coli are very toxic). But it is certainly true that an individual who has high Gram-positive stools can by the autogenous mixed or B. coli instillations quickly have the running proportion between the Gram-negatives and positives raised to a proportion equivalent to normal, this being due to a raising in the B. coli and also to a diminution in the putrefactive Gram-positives as the first become more numerous. With this more equal proportion between the two types of organisms, the conjugate sulphate of the urine diminishes and the cases make substantial improvement in the general body. Whether this raising of the Gram negatives is only due to the B. coli or only to the B. lactosus aërogenes (both being antitoxic to other bacteria), or to both together, is not always possible of determination, since both are much alike in their morphology and are Gram-negative in character. But the cultural methods of distinguishing these two forms from each other and the results obtained when only the B. coli were used in the injections

incline me to believe that these disorders are due to a shortage or inactivity of the B. coli, and that the latter are the most powerful agents in the human alimentary canal against the development of putrefactive conditions, and, that while outside of the intestine they are destructive and pyogenic, inside of the canal they are welcome hosts. As regards the permanency of the benefit brought about it is apparent that about half of the cases which do not respond to simple treatments clear up inside of from one to three months on this treatment, but that the other half may not remain substantially benefited even when the instillations are kept up for longer periods. These latter show relapses when the instillations have been stopped for a week or more, quickly responding again when the injections are reëstablished and some eventually clear up. It is probable that in the relapsing cases some permanent anatomical mischief preventing the establishment of a normal bacterial intestinal condition is present, which is either the cause of the development of the condition in the first instance and then its prolongation, or that there is present some anatomical or permanent functional change affecting normal secretions and motility of the digestive canal in asthenic ways. All cases of putrefactive conditions should first be treated by the routine methods of treatment (diet, hygiene, tonics, etc.) before instituting the instillations. If no benefit is noted on the B. coli alone the B. lactosus aërogenes may also be added to them, the two grown together in the single media, and these tried for a length of time. And if after these no sustained or apparent benefit is achieved, then we have present some anatomic and permanent complication affecting the function of the gut, and the best we can hope for is a resort to surgery in some of the cases, or a longer interval continuation of instillations of whatever form of culture has shown the best results in that particular case."

After numberless attempts to administer rectally the innocent forms of coli vaccine, I have come to the conclusion that not only do they act in a beneficial way by inhibiting the processes of fermentation and putrefaction by such effect as they exert upon the bacteria present in the intestinal canal but they produce a leucocytosis or a stimulation of leucocytes in the walls of the intestine with the generation of more or less of an antibody formation which acts as a means to elevate resistance against bacteria in a general way. I cannot explain the results accomplished along any other line than that there must be this leucocytic antitoxic body formation, because the individuals remain permanently well and are singularly free from infections of all kinds for some years. It may be, after all, that the

results accomplished by my method are distinctly along the vaccine immunity line, and not along the line of increasing the number of colon bacilli in the intestinal canal, or any local effect of that sort.

The choice between the two methods—namely, subcutaneous of dead bacteria which have been killed by heat (and never by means of any phenol substance) or the viable autogenous rectally instilled, is a matter according to the individual case. After a length of time one instinctively can draw distinctions as to which is the wiser method to pursue, whether by vaccination on the one hand or for antagonistic effect on the other. This is somewhat of an art and cannot be described in words. There are some instances where it is wise to use both methods at the same time, but this is not a good practice because it is not possible to state which one of the methods brought about the beneficial results. It is better to use one, then the other or, better yet, to decide on which is the best one to use according to the individual case. In a general way, infections of the intestinal content are best controlled by means of the rectal administration while those of the true toxemia wherein there is invasion and infection of the mucosa or tissues of the body, by the subcutaneous. Many cases have both an infection of the content and infection of the tissues and therefore require both methods of attack eventually.

The method of taking innocent forms of bacteria by mouth, the subcutaneous immunity vaccine method, and the rectal instillation of B. coli has failed me at times. This is sometimes due to fault in the selection of the case and at other times due to improper vaccine employed—occasionally to improper laboratory procedure in estimating the pathogenic forms present in the individual. Ofttimes it is necessary to re-examine the case, go over the stools carefully again, particularly from a bacteriological standpoint, and do a restudy of the sedimentary field. It is not uncommon that a conclusion which was drawn in the first instance was distinctly different from the conclusion that is drawn in the second and third, and so on. The colon bacilli are great complicators of the work and not uncommonly an underlying or overlying bacteriology is very much more important in the production of the symptoms than the presence of the B. coli. In these instances no results, or only very mild ones, would be brought about by the use of the coli vaccine, and as my work multiplied the number of cases in which other vaccines were more desirable constantly increased until now over 2000 cases have been treated with other than the B. coli.

It is not advisable to give a long dissertation on the *pros* and *cons* on the elaborated vaccine side of the subject, particularly in the matter

of ideally selecting the bacteria for the case. This can only be accomplished by careful laboratory study. The following is the plan of the work I now follow when I employ the vaccine immunity method in the case, giving the infecting organisms, and the two methods of administration of the vaccine. A single bacterium is always employed in the vaccine used.

BASSLER'S BACTERIAL TREATMENTS IN PRIMARY TOXEMIAS.

Rectal and Subcutaneous Routes.

Vaccine Immunity Methods.

SACCHARO-BUTYRIC	B. aërogenes capsulatus (rectal) (skin; rarely) Gram-positive diplococci (rectal; rarely) Gram-positive single cocci (rectal) B. bifidus (rectal; rarely) B. putrificus (rectal; rarely)
INDOLIC	B. coli communis (rectal) (skin) Identification of which of the 17 varieties, and that one used. B. mysentericus (rectal) B. liquefaciens (rectal) B. proteus Gram-negative streptococci (skin) Staphylococci (skin)
MIXED	Combinations of above according to predomination of fermentation or putrefaction, and types of organism. The rectal method is used here altogether, and effort is made to get reactions and a leucocytosis of from 10 to 20 thousand within eight hours after the injections.

After all that has been said in connection with the diet, general care and vaccines, there still can be found instances where there is no result worth while, either such as can be proven in the laboratory or results which the individual shows in a state of improved health. In these instances it has been my custom, after an extensive study of bacterial antagonisms, to use the following method of administering bacteria, all the work then being done either by rectal administration or administration through the duodenal tube, usually the first. After seven years experience in the study of bacterial antagonisms to meet such cases as fail to respond to the vaccine immunity plan, the following represents the plan I now follow. It must be remembered here, however, that the results may not be as substantial as those accomplished by means of the vaccine immunity method:

BASSLER'S BACTERIAL TREATMENTS IN PRIMARY TOXEMIAS.

Rectal and Subcutaneous Routes.

Bacterial Antagonism Methods.

SACCHARO-BUTYRIC (Heavy protein diet)	B. aërogenes capsulatus Gram-positive diplococci Gram-positive single cocci B. bifidus	B. coli (many different strains and perhaps collected from different sources). For the first two the *a*, for the second two the *b* strains are best.
INDOLIC (Low protein and high carbohydrate and hydrocarbon diet).	B. coli B. mysentericus Gram-negative streptococci Gram-negative staphylococci	B. acidophilus B. bulgarius B. lactis aërogenes G. P. diplococci G. P. cocci
	B. proteus vulgaris (B. Welch) B. cloaca (B. coli, polyvalent strains) B. pyocyaneus (B. coli, *a* strains) B. putrificus (B. coli, *b* strains)	
MIXED (Least possible amounts of food, no cheese, peelings of fruits—mostly boiled foods).	No action on antagonisms possible by rectal or subcutaneous methods excepting when a predominant type of bacteria is present.	

The difference between the *a* and *b* strains of B. coli is that the *a* does not produce gas in saccharose; the *b* does. The effects are the same on all the other sugars and on the coagulation of milk.

Not a few of the cases have been treated along a line which I have designated as biochemical alteration. In these instances, as in the vaccine method, the infecting bacterium is employed. Different from the vaccine method in which the organism employed is gained in as pure culture as possible in the shortest time after the stool specimen is on hand, by the biochemic alteration method the vaccine used is that in which the organism has been grown in successive subcultures, these averaging between four and six. According to the organism, the media is changed, sometimes at each inoculation, the idea being to change it both morphologically and in chemical ways. This often robs it of its specificity and toxicity, and at the same time it answers for vaccine effect on the organism infecting the host. It is only possible by the study of the individual case and then perhaps after more or less instillation experience to decide whether the biochemic alteration

would be the best to employ either all the way through the bacterial treatment time or in a part of it. The list of this method is the following:

BASSLER'S BACTERIAL TREATMENTS IN PRIMARY TOXEMIAS.

Rectal Route.

Biochemical Alterations.

Occasionally infecting bacteria can be changed biochemically by growing under different media and these used in the effort to substitute those present in the body. Successful examples of this have been found in cases of infections with the B. coli, aërogenes capsulatus, mysentericus, and putrificus.

SURGICAL TREATMENT OF INTESTINAL TOXEMIA (Stasis).

Watching the number of baneful results that have been accomplished by surgeons who have followed the propaganda of Lane and others, I am pleased that I have not worshiped at the altar of the surgical treatment of intestinal toxemia for the purpose of changing the biology of the intestinal canal. I am satisfied that the surgical therapy of drainage, (which would have to do with pus, urine, etc.). or the removal of diseased tissue are surgical fundamentals that should be continued. But, these conditions of toxemia are a biologic state and one cannot change the biology of the intestinal canal in satisfactory ways by changing the fecal current. Deaver was perfectly right about that. To remove an appendix; or to relieve a kink which is causing a distinct obstruction, may in individual instances be a justifiable surgical procedure. There are a few cases of reconstruction of the right side of the colon in which it may also be indicated. But the removal of the colon, or to do an anastomosis where there is not a distinct intestinal obstruction is bad surgical procedure, and it is only necessary to follow a number of these cases which have been operated upon by enthusiastic surgeons to prove this. It is unfortunate that the biology of this subject is not as well understood by surgeons as it should be, but it is plain to me that of late they are not engaging in as much exploitation in this field as several years ago, evidently because they did not accomplish results which were substantial or none at all or that the rate of mortality from excision was too high. It is not uncommon to see patients improve for a while after such surgical procedure, but usually in the course of six months or a year, almost always by the latter period, they are quite as bad as they were before, simply because their intestinal toxemia still exists. And it is not uncommon, even when the entire colon has been removed, to see a condition of affairs which is worse than the state of the individual before the operation was performed. The more experience I

have, the fewer the operations of questionable sorts are suggested, and when the case is simply one of an intestinal toxemia, however much pathology there may be in the right half of the colon, it is never considered surgical, but always treated medically. In a word, for the treatment of intestinal toxemia in all stages, surgical procedure is not indicated; if a definite obstruction exists, *yes*, but for the ordinary case, *no*. There may be a resulting pathology in the appendix, gall-bladder, etc., that requires surgery, but for the toxemia or such stasis that is not due to definite obstruction, never.

References.

[1] Bouchard: Lecons sur les Autointoxications, etc., Paris, 1887.

[2] Metchnikoff: "Scientifically Soured Milk; Its Influence on Arresting Intestinal Putrefaction," etc., Paris, 1907; "The Prolongation of Life," etc., New York and London, 1908.

[3] Herter: "Bacterial Infections of the Digestive Tract."

[4] Lane: "Chronic Intestinal Stasis," Trans. of the Am. Surg. Assn., 1909, p. 23; "Operative Treatment of Chronic Constipation." London, 1909; "The Kink of the Ileum in Chronic Intestinal Stasis," London, 1910.

[5] Watson: "Chronic Intestinal Stasis," The Clin. Jour., London, June 30, 1915.

[6] Mayo, Charles H.: "Resection of the First Portion of the Large Intestine," etc., Jour. Am. Med. Assn., Aug. 8, 1914, p. 446; "Removal of the Right Colon," etc., Jour. Am. Med. Assn., Sept. 9, 1916, p. 779.

[7] Kellogg: "Incompetency of the Ileocecal Valve," 1913; "Surgery of the Ileocecal Valve," Surg., Gyn., and Obstet., Nov. 13, 1913, p. 563.

[8] Martin, Edward: Paper read at Boston Medical Library, Nov. 19, 1913; "Therapeutic Answer to Chronic Right Ileac Pain."

[9] Keith: Brit. Jour. Surg., 1914-1915, ii, p. 576.

[10] Combe, A.: "Intestinal Autointoxication."

[11] Lecoq: Biol. medic., 111, p. 368.

[12] Combe, A.: "Intestinal Autointoxication."

[13] Herter, Christian A.: "The Common Bacterial Infections of the Digestive Tract and the Intoxications Arising Therefrom," Jour. Am. Med. Assn., March 23, 1907.

[14] Distaso, A.: Centralbl. f. Bakteriol., etc., Abt. 1, Orig., Bd. 62, H. 6, p. 433.

[15] Cohendy, M.: Annales de l'Institut Pasteur, T. xxvi, p. 106.

[16] Distaso, A.: "The Intestinal Flora," The Lancet, Feb. 24, 1912.

[17] Distaso: These tubes show the same action as our B. cellulosæ disagregans in pure culture. Societe de Biologie, 1911.

[18] Eppinger, H. and Guttmann, J.: Ztschr. f. klin. Med., vol. lxxviii, Nos. 5 and 6.

[19] Herter, C. A.: "Indolaceturia," Jour. Am. Med. Assn., June 13, 1908.

[20] Nencki and Sieber: Jour. f. prakt. Chem., 1882, xxvi, 333.

[21] Hopkins and Cole: "The Constitution of Tryptophane and the Action of Bacteria on it," Jour. Physiol., 1903, xxix, 451.

[22] ELLINGER: "Ueber die Constitution der indol Gruppe im Eiweiss (Synthese der sogen, Skatolcarbonsaüre) und edie Quelle der Kynurensaure," Ber. d. deutsch. chem. Gesellsch, May 4, 1906; No. 7, p. 1802.

[23] PORCHET and HERVIEUX: "Untersuchungen über das Skatol," Ztschr. f. physiol. Chem., 1905, xlv, 486.

[24] KENDALL, ARTHUR I.: "The Relation of Bacterial Metabolism to Certain Foods."

[25] VON NOORDEN: Jour. Am. Med. Assn., Jan. 11, 1913.

[26] ROSS: Archives of Internal Medicine.

[27] ALVAREZ: Jour. Am. Med. Assn., Dec. 15, 1917.

[28] BASSLER: Medical Record, March 7, 1914.

[29] ROSS: Archives of Internal Medicine.

[30] ASKENSTEDT: New York Med. Jour., Oct. 9, 1909.

[31] HIROSE: Deut. Med. Woch., July 25, 1912.

[32] BOUCHARD: Lecons sur les autointoxications dans les maladies, Paris, 1887.

[33] BAUMANN: Ztschr. f. physiol. Chem., 1886, x, 123.

[34] SUCKSDORFF: Arch. f. Hyg., 1886, iv, 355.

[35] KUMAWAGA: Virchows Arch. f. path. Anat., 1888, cxiii, 134.

[36] SALKOWSKI: Virchows Arch. f. path. Anat., 1889, cxv, 339.

[37] STERN: Ztschr. f. Hyg., 1892, xii, 88.

[38] MIECZKOWSKI: Mitt. a. d. Grenzgeb. d. Med. u. Chir., 1902, ix, 405.

[39] ASSMANN: Ztschr. f. Tiermed., 1911, xv, 122, 264, 352.

[40] KLIMMER: Ztschr. f. Tiermed., 1900, iv, No. 4.

[41] STRASBERGER: Ztschr. f. klin. Med., 1903, xlviii, 491.

[42] FEIGEN: Inaug. Diss., Bonn, 1908, p. 39.

[43] FRIEDENWALD and LEITZ: Am. Jour. Med. Sc., 1909, cxxxviii, N. S., 653.

[44] BERGER and TSUCHIYA: Ztschr. f. exper. Path. u. Therap., 1910, vii, 437.

[45] STEELE: Jour Med. Research, 1908, xiii, N. S., 93.

[46] MIECZKOWSKI: Mitt. a. d. Grenzgeb. d.Med. u. Chir., 1902, ix, 405.

[47] SUCKSDORFF: Arch. f. Hygiene, 1886, iv.

[48] STERNBERG: Text-book of Bacteriology, 1896, p. 201.

[49] HARRIS: Jour. Am. Med. Assn., Oct. 12, 1912.

[50] WRIGHT and DOUGLASS: Lancet, Sept. 17, 1910.

[51] CHVOSTEK: Wien. klin. Wchnschr., April, 1908, xxi, 453.

[52] TURCK, FENTON B.: International Clinics, 1911, xi, 45.

[53] SATTERLEE, G. REESE: Jour. Am. Med. Assn., Dec. 9, 1916.

[54] DEAVER, JOHN: Appendicitis, Its Diagnosis and Treatment, 1905.

[55] BASSLER: Medical Record, Sept. 24, 1910.

CHAPTER VIII.

Chronic Excessive Intestinal Toxemia.

(Continued.)

CHRONIC INTESTINAL STASIS.
(Non-obstructive.)

In presenting the author's opinion of what chronic intestinal stasis is he desires first to mention what have been described as chronic intestinal stases but which in his opinion are not:

Constipation	Intestinal toxemias (any type)	Splanchnoptosis
Gastro-duodenal ulcer and sequelæ	Pyloric obstruction	Duodenal adhesions
Mesenteric ileus	Adhesions at the duodeno-jejunal angle	Lane's kink
Incompetent ileocecal valve	Chronic appendicitis and appendicular adhesions	Mobile and dilated cecum
Jackson's membrane	Adhesions at the hepatic flexure of the colon	Prolapsed colon
Adhesions at the splenic flexure of the colon	Adhesions of the sigmoid	Angulation at the recto-sigmoid junction
Pericolonic adhesions (anywhere)	Intestinal venous stasis	Flatulence
Cystic degeneration of the breast	Intestinal obstruction (any cause)	Non-rotation of the colon
Headaches, dizziness, depression and weakness		

It may now be asked, if the above are not symptoms of intestinal stasis, what is intestinal stasis? In my opinion intestinal stasis is a symptom, and is no more characteristic of an entity than is cough a symptom of pneumonia, or pain in the chest one of thoracic aneurysm. In any of the above conditions intestinal stasis may be present in fact commonly is, but intestinal stasis is a symptom, and usually is secondary to or a part of most of the above mentioned conditions. Chronic intestinal stasis is an abnormal slowing of the

transit through the intestinal canal, usually in the lower part of the ileum or in the right or transverse colon, and is due either to obstructive causes, when the diagnosis should be that of intestinal obstruction, or, to an etiology the nature of which may be described as follows:

There first takes place an infection of the intestinal content with a resorption of toxic bodies. Commonly also present is an infection of the mucosa with a degeneration in the ganglia of the Auerbach and

Fig. 49.—Pathology on right colon due to intestinal toxemia. Cecum. Showing normal mucous membrane and caput cecum above ileocecal valve, chronic inflammation of the ileocecal valve (which was firm and closed when tissue was removed but is shown relaxed because the specimen was one day old when photographed), areas of hemorrhage and ulceration in the cecum beyond the valve, the mucous membrane atrophic and the gut wall thinned.

Meissner plexuses, and more or less absorption of the fibers which run to the ganglia or come from the various cells. This condition causes an intestinal stasis and is the most common cause.

The difficulties in the subject have been multiplied because under the term intestinal stasis have been included numerous conditions in the abdomen in which stasis was present. This has been brought about largely by the propaganda of Lane and quick conclusions on X-ray methods of examination, operation being performed upon

the basis of a stasis (the case being designated as intestinal stasis) and not designated according to the pathologies which were found in which the intestinal stasis was an accompanying or secondary condition. It is these facts that have brought about confusion in the subject, which was and is unnecessary.

In not a few cases of chronic excessive intestinal toxemia, particularly in the indolic and mixed types, when the condition has existed

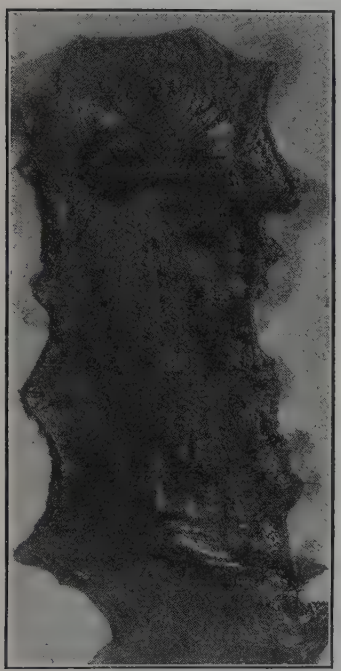


Fig. 50.—Same as Fig. 49. More of the gut, showing an extension of the chronic condition in the ascending colon.

Fig. 51.—Same as Figs. 49 and 50.—Still more of the gut, showing a gradual transition of the pathology into normal hepatic flexure and transverse colon beyond, also showing the places from which tissue was cut for sections, these being numbered according to illustrations of section shown.

for a long time and has been neglected, pathology takes place in the mucous membrane. This is due to resorption of toxic material from the interior of the gut. The first change noticed is usually a swelling of the epithelium giving it a glossy thickened appearance, and somewhat smoother than normal. In the course of time a necrosis brought about by a round-cell infiltration in the mucosa causes an absorption of

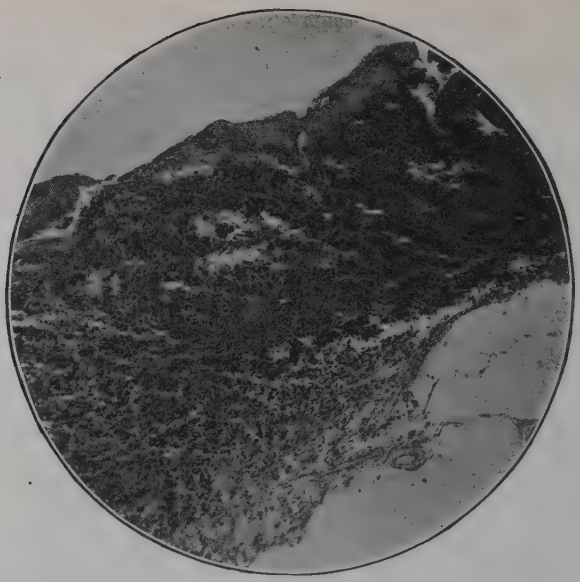


Fig. 52.—This specimen shows degeneration and necrosis in the mucosa, which is more particularly noticeable in the lymphadenoid tissue and glands, the latter not showing in this photomicrograph, which shows a degeneration and necrotic area in the lymphadenoid tissue. There are no marked changes in the submucosa. ×145.. (No. 1, Fig. 51.)

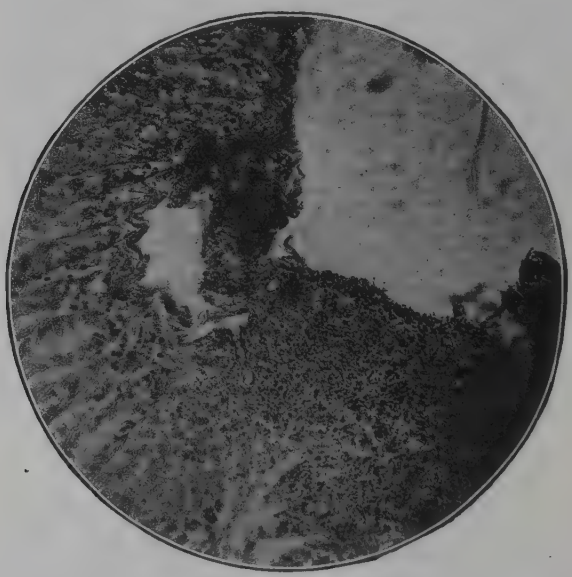


Fig. 53.—This specimen shows extensive degenerative and necrotic changes in the mucosa, among the glands and lymphadenoid tissue. Some of the glands are denuded of epithelium. There are some slight degenerative changes in the submucosa, but these are not nearly so marked as in the mucosa. Could detect no signs of hemorrhage. ×145. (No. 2, Fig. 51.)

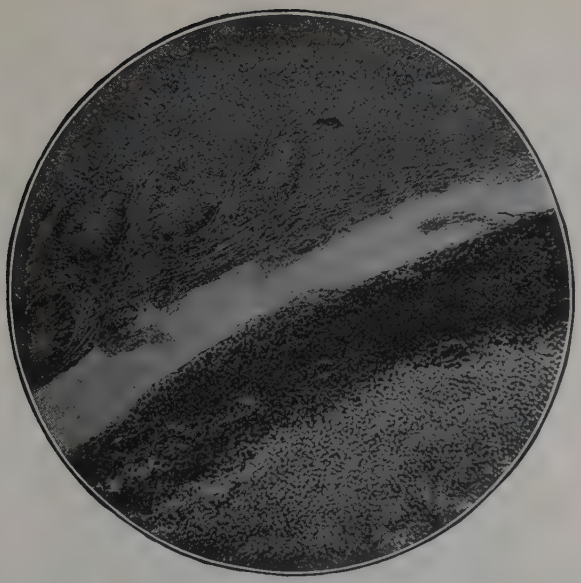


Fig. 54.—Like 52 and 53, this shows considerable degeneration and necrosis in the mucosa among the glands and lymph nodules, some of the former even showing desquamation of glandular tissue. There are also some slight degenerative changes in the submucosa (not shown in this picture). The specimen is truly atonic. ×145. (No. 3, Fig. 51.)

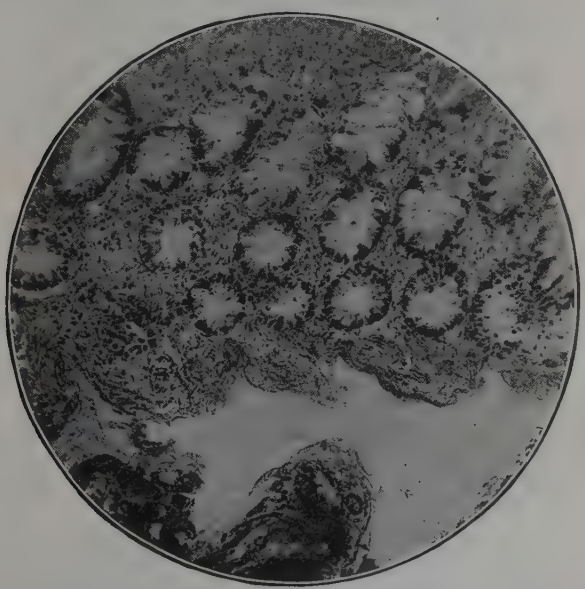


Fig. 55.—This specimen is more nearly normal than any of those shown (Figs. 52, 53, 54), but in places it still shows degeneration and necrosis in the mucosa among the glands and lymphoid tissue. All such foci in the stained specimen are of much paler color than in the normal areas. ×145. (No. 4, Fig. 51.)

the elements in the mucous membrane, mainly the cellular, which is replaced by a stroma, sometimes very homogeneous in character. Sections of such mucous membrane may show a complete absence of tubular cells, the mucous membrane being injected with degenerative and abnormal material noticeable mainly in the lymphadenoid tissue and glands. This degeneration and necrosis extends causing a des-

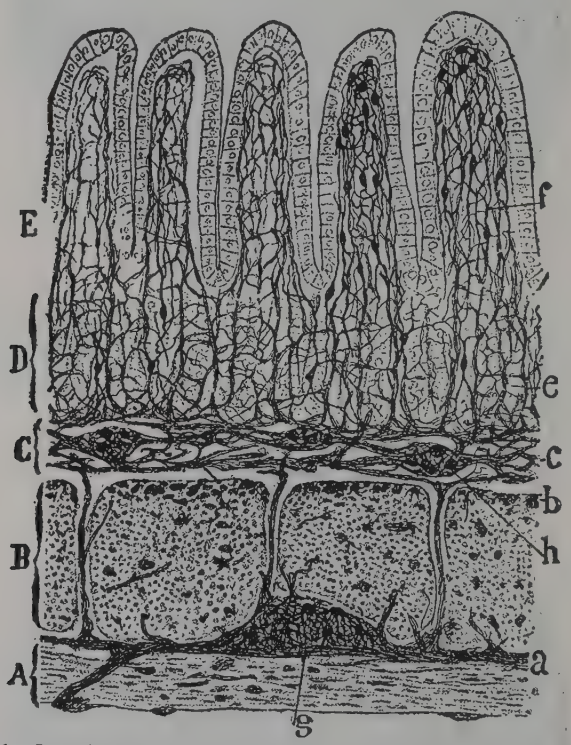


Fig. 56.—Longitudinal section from the small intestine of a guinea-pig —*Golgi* method. (This picture is partly schematic to show the general effect of the plexus and ganglions.) *A*, Layer of longitudinal muscular fibers. *B*, Layer of circular muscular fibers. *C*, Connecting submucus tissue with the plexus of Meissner and its ganglions. *D*, Layer of glands of Leiberkühn. *E*, Villi. *a*, Plexus of Auerbach. *b*, Deep muscular plexus cut transversely. *c*, Balustrades of the plexus of Meissner. *e*, Balustrades of the periglandular plexus. *f*, Intravillus plexus. *g*, Ganglion of the plexus of Auerbach.

quamation of the cellular tissue, finally the destruction of the glandular tissue, and perhaps some slight degenerative changes in the submucosa. The process is usually met with in the lower ileum and mainly in the right colon, although it may extend all the way from the lower ileum to practically the dome of the rectum.

Fig. 57.—Normal ganglion of Auerbach plexus. (*Ehrlich* method.)

Fig. 58.—Ganglion from Auerbach plexus in a case of chronic toxemia, showing shrunken ganglia and fine granular degeneration of the protoplasm.

Viewing such mucous membrane it looks dry and thin, generally wrinkled, and here and there are hemorrhagic areas commonly with small ulcerations. In my experience they are never met with in the caput of the colon, but correspond to the course of the transit of food through the gut, the ileocecal valve area being tremendously injected or congested, but showing nothing more than a simple hyaline swelling in the superficial layers of the mucous membrane. As the condition continues there is a pathologic change in the ganglionic cells of the Auerbach and Meissner plexus in which the ganglionic cells

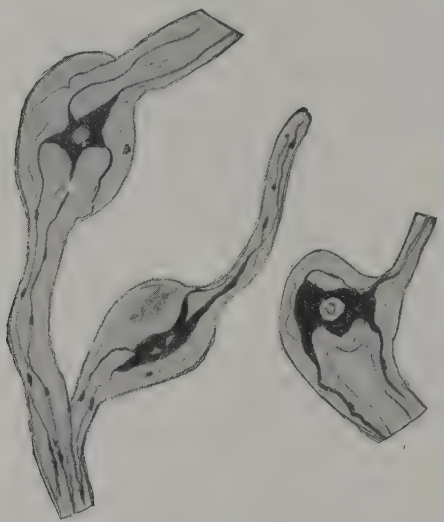


Fig. 59.—Cells of the Meissner plexus, normal human being. (*Ehrlich* method.)

become degenerated at the outer aspects, the fine granular degeneration commonly extending to almost the center of the cell. This degeneration is in the protoplasm of the cell interfering with its function, which function is further interfered with by the afferent and efferent branches going to and from the ganglionic cells withering up in some instances, others becoming absolutely detached, absorbed, and usually in all instances, the total number being tremendously diminished.

Manifestly such a condition interfering definitely with the enervation of the muscular fibers, mainly the circular, produces what is called intestinal stasis. Such a pathology may be secondary to conditions such as bands, kinks, ptosis, and others, although it is rarely possible to state that the condition is secondary even though these

anatomical defects are noted, because commonly these anatomical defects are themselves secondary to an original chronic intestinal toxemia which has not been discovered, and no symptoms had arisen until definite stasis existed when they may be those of the stasis entirely. It is not warranted or fair to make a diagnosis of chronic intestinal stasis when a diseased appendix, or some other pathologic condition is present in the abdomen with stasis as an accompaniment, and after an operation had been performed to describe the case as one of stasis. If the medical profession will keep this designation of intestinal stasis in mind, there will be no confusion as to where the term chronic intestinal stasis should be limited. It should

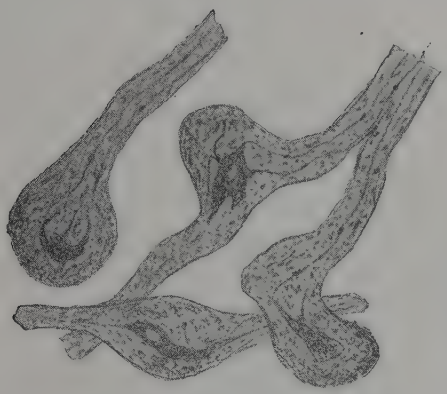


Fig. 60.—Cells of Meissner plexus in a case of chronic intestinal toxemia; cells markedly shrunken and degenerated. Poor staining of nucleus.

not be used to describe many conditions the definite pathology of which (in fact, the treatment of also) are well-known and have been in the realms of medicine and surgery long before Lane's propaganda. To describe operative procedures for conditions which have caused obstruction, either by interference with the dynamics of the circular fibers or by actually constricting or anchoring a gut, as intestinal stasis is like the tail wagging the dog.

Symptoms.—The symptomatology of chronic intestinal stasis therefore is entirely a Roentgenological matter. It comprises the delay in the small intestine usually noted at the eighth hour observation after the ingestion of the opaque meal, when it should be almost entirely in the right colon; its delay in the right colon, sometimes extending to as long as thirty-six or forty-eight hours; its delay in the transverse colon, sometimes a matter of two or three days. But the delay in the left colon had best not be considered in connection with

this diagnosis, because actual stasis due to degeneration of the ganglionic cells is not as commonly found in the left colon as in the areas above.

It must be remembered that when intestinal stasis has been diagnosed that one is dealing merely with a symptom; that the diagnosis of the actual condition has still to be made, which in most instances is a chronic excessive intestinal toxemia, although it may present

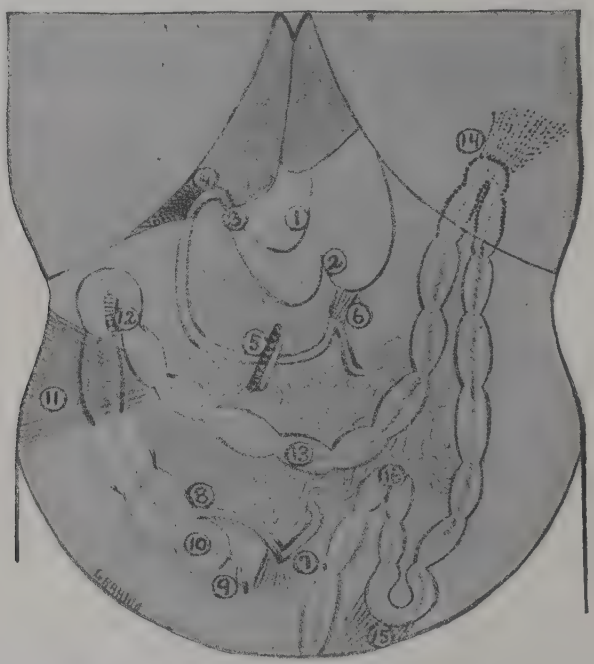


Fig. 61.—Diagram showing causes of gastro-intestinal stasis. (*Graham.*) 1, Prolapsed stomach. 2, Hour-glass stomach. 3, Pyloric obstruction. 4, Adhesions at the duodenum. 5, Mesenteric ileus. 6, Adhesions at duodenojejunal angle. 7, Lane's kink. 8, Incompetent ileocecal valve. 9, Chronic appendicitis. 10, Mobile and dilated cecum. 11, Jackson's membrane. 12, Adhesions at the hepatic flexure of the colon. 13, Prolapsed colon. 14, Adhesions at the splenic flexure of the colon. 15, Adhesions at the sigmoid. 16, Angulation at the rectosigmoid junction. (This is offered by the author, not as a total of the causes, but to show the common sites of stasis.)

any one of the conditions mentioned above in connection with what intestinal stasis actually is not, but only secondary to.

Treatment—The treatment for chronic intestinal stasis is the treatment of the original condition, whatever this may be, or whatever combinations of them may be present. These are mentioned in

connection with the various subjects and the author considers it out of place to describe any method of treatment for intestinal stasis as such, since the treatments for it are the treatments for the various conditions that have brought the stasis about.

INCOMPETENCY OF THE ILEOCECAL VALVE.

It certainly was not later than 1910 that my attention was called to incompetent ileocecal valves by Cole of New York. At that time he had been studying the entrance of bismuth clysma from the right colon into the lower ileum, and was much interested in the subject.

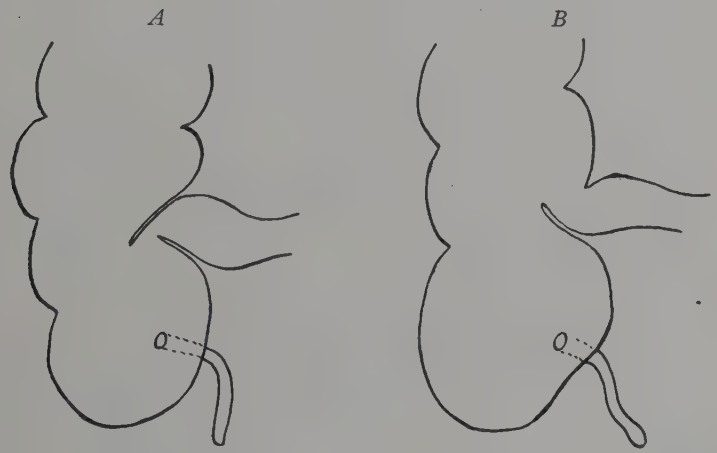


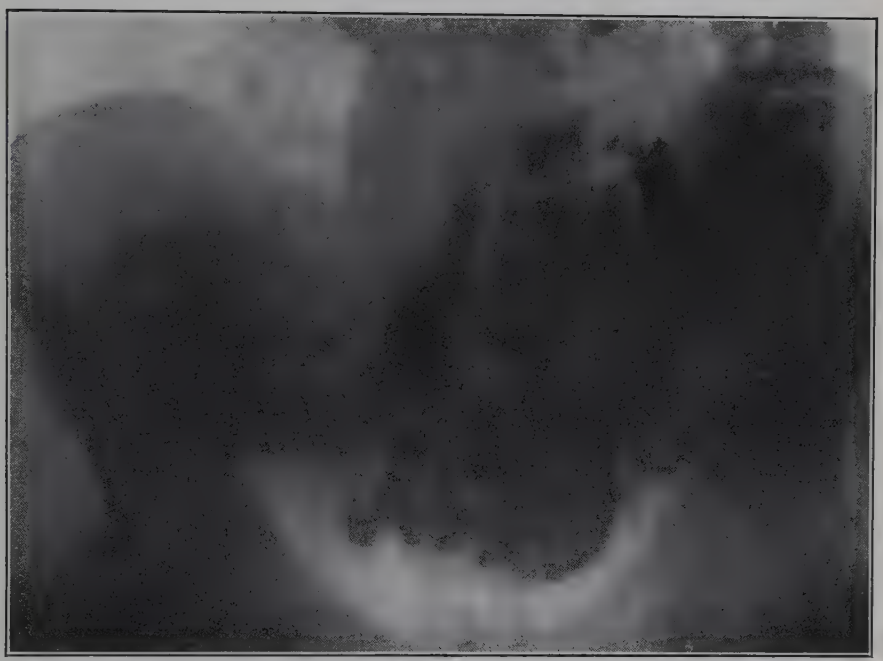
Fig. 62.—*A*, Normal ileocecal valve. *B*, Incompetent ileocecal valve.

My interest in the matter began then and during the course of two or three years I noted there were numberless instances of people who had been examined in my X-ray department who had incompetent ileocecal valves without symptoms that could be ascribed to it. In 1913 Kellogg[1] drew attention to the incompetency of the ileocecal valve, disorders arising from this condition, and a method of treatment. His conclusion was that in complete incompetency of the valve the passage of undigested material could take place from the small intestine into the colon, causing an irritation and diarrhea. He suggested that when the passage from the small intestine into the large was too rapid, distention of the colon, stasis, putrefactive processes, colitis, pericolitis or appendicitis might result. The accumulated material drawing upon the root of the mesentery could produce an obstruction at the junction of the duodenum and jejunum and cause duodenal and gastric stasis. Appendicitis and various troubles

attributable to adhesions about the ileocecal valve region were a necessary consequence of incompetency of the valve, and this incompetency interfered with the movement of gases, causing flatulence, which he claimed to be characteristic of complete incompetency of the valve, as he observed in 35 per cent. of the cases studied. Constipation was another of the consequences of incompetency of the ileocecal valve, and lastly, incompetency of the valve gave rise to the condition of intestinal intoxication, such as has been pointed out by Schmidt. He mentioned certain medical measures of control of these cases, and finally suggested a surgical procedure as a radical cure for incompetency of the ileocecal valve, claiming great benefit by it.

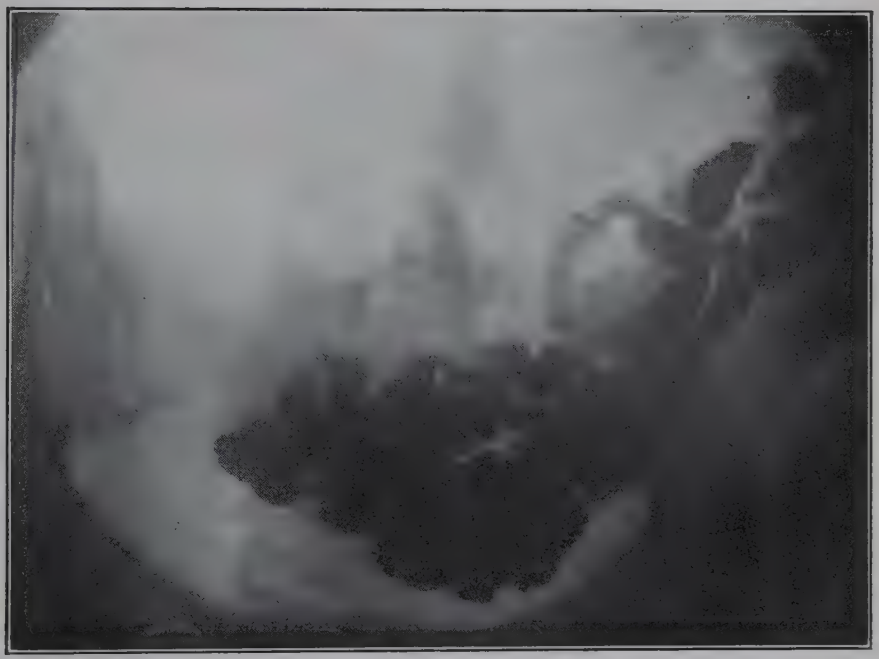
After an intensive study of this subject I have come to the conclusion that incompetency of the ileocecal valve is not a subject of much importance in medicine. It is present in at least one out of every six or eight persons, depending upon the X-ray technique employed for its observation. Take for instance a normal person who has been starved for awhile and a clysma of considerable amount given; a small amount of barium suspension can always be found in the lower end of the ileum. During conditions of starvation for whatever purpose, the valve becomes incompetent, and there are not a few individuals having incompetent valves who are perfectly normal. In my opinion an incompetent ileocecal valve does no harm from the standpoint of the fluids from the small intestine rushing rather faster into the large. In watching valves, as I have for a long time, it is plain to me that if the descending and transverse colon are clear, foods rush through the most competent valves just about as quickly as they can and do; not going through in small jets as from the pylorus into the duodenum. I am satisfied that a slight degree of stasis may occur as a direct consequence of a markedly incompetent valve, but I think that the degree of stasis occurring is of such slight extent that it has no marked significance, certainly not in a surgical way. As to whether the incompetency of the valve can lead to the production of pericolitic adhesions and so on, is a question which I doubt if anyone can prove. The point to remember is that incompetency of the ileocecal valve is not a primary condition. It is secondary to an intestinal toxemia in which there has been a pathology in the gut wall, or especially a pathology in the sympathetic fibers of the Meissner and Auerbach plexuses, perhaps the extra enteric fibers and ganglia as well. This is a primary condition and the relaxation of the valve is quite secondary. I do not believe, for instance, that incompetency of the valve by permitting a larger accumulation in the right colon produces an obstruction of the duodenum and jejunum and a gastric stasis. In the number of cases

PLATE XIII



Marked ileal stasis eighth hour after ingestion. In this case there is general lack of tone and function in the small intestine without anatomical conditions. (X-ray by author.)

PLATE XIV



Marked ileal stasis due to chronically diseased appendix.
Eight hours after ingestion. (X-ray by author.)

PLATE XV

General colonic stasis. Thirty-six hours after ingestion. Due to ptosis and general colitis. (X-ray by author.)

PLATE XVI

Eight-hour gastric stasis due to adhesions from the gall-bladder. Colon fairly well filled. (X-ray by author.)

PLATE XVII

Incompetency of the ileocecal valve. The fluid contents could be pushed from the ileum into the colon and back again by hands on the abdomen. (X-ray by author.)

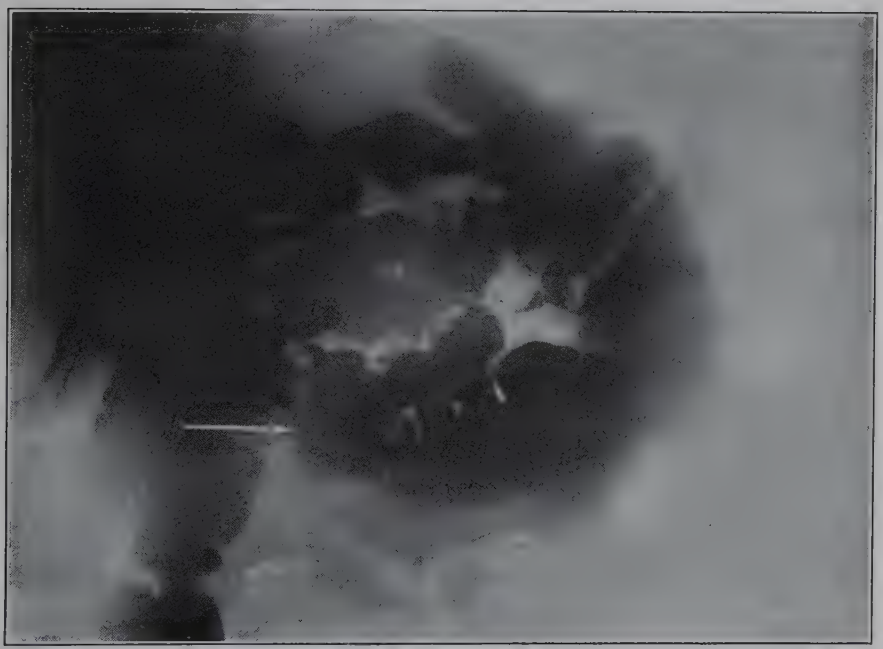
PLATE XVIII

Adhesions in the vicinity of the appendix due to chronic disease of the organ. (X-ray by author.)

PLATE XIX

Adhesions near the hepatic flexure. Arrow pointing to site. Intestinal obstruction. Cause of the adhesions, chronic intestinal toxemia with resulting peritoneal infection. (X-ray by author.)

PLATE XX



Adhesion-bearing area in distal transverse colon. Pericolonic from intestinal toxemia. (X-ray by author.)

of incompetency of the valve that I have observed, I have yet to see the first case of distinct gastric stasis as a consequence of it. Incompetency in my opinion is secondary to infection of the gut content or of the gut wall and this infection is not a consequent condition to any ileocecal valve incompetency. Perhaps it is of some importance in connection with the presence of gas which may be allowed to go through an incompetent valve into the lower ileum, but this is a matter easily corrected in practically all instances by attention to the colon further down. That incompetency of the ileocecal valve gives rise to conditions known as intestinal intoxication I do not believe at all. It is quite the other way—the incompetency of the valve is secondary to the intestinal condition. Such cases as I have seen in which primary surgery and repair of the ileocecal valve have been done are just the same in the course of a few months after the moral effect of the operation has subsided. Toxemia goes on as before and therefore the symptoms return. There seems, however, to be a slight degree of benefit in some instances, but they are very few considering the large number of incompetent valves one meets in clinical work, and the number that have been operated upon.

Treatment.—It is essential when incompetency of the ileocecal valve is noted to figure as to how much or many, if any, of the symptoms are due to the incompetency of the valve itself. Generally on the closest analysis, in nearly every instance, it will be concluded that the incompetency of the valve does not produce symptoms by itself and certainly is not a cause of intestinal toxemia. Such degrees of stasis as may exist are generally best handled along medical lines directed to the underlying toxemia that exists. It is important in these instances to keep the colon cleared by the use of an anti-constipation diet and by such means as empty the left side, particularly the transverse colon. In these instances the use of suprarenal tablets or an adrenaline solution is of some value in keeping a valve competent. It must be remembered in all of the cases that generally there is dilatation, and that it is the treatment of the underlying conditions that are the most successful in handling the incompetent valve. In a few cases, perhaps, if the abdomen must be entered for the removal of a diseased appendix, binding adhesions, or something of that sort, a valve may be repaired by the method suggested by Kellogg. To do a primary operation upon an incompetent valve in the hope that it will correct the conditions that are supposed to be caused by it, is in my opinion a mistake.

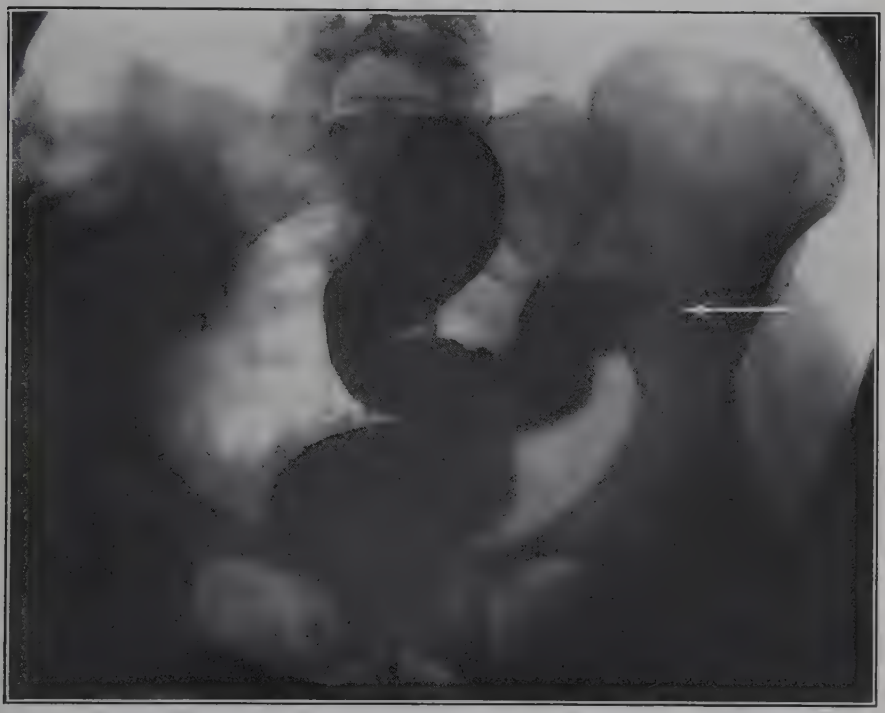
PERICOLONIC ADHESIONS.
(Inflammatory.)

Pericolonic adhesions are due to bacteria of the content of the gut having passed through the coats of the wall of the intestine, entering the peritoneal surface, causing a slight degree of plastic peritonitis, which when formed at the site of contact with a fresh surface of another portion of the intestine, causes a corresponding process there, providing the parts lie in apposition long enough, and this results in an adhesion. These adhesions are met with all the way from the ileocecal valve region to the brim of the pelvis on the left side. They are quite common in the ascending colon region and ofttimes have been ascribed to the presence of a diseased appendix. It is possible that a diseased appendix may by virtue of its lowered resistance permit the transit of bacteria through the walls and the bacteria gaining the peritoneal surface may, by migration or transplantation to approximate portions of the gut, cause adhesions to form. In most of the instances, however, the adhesions are undoubtedly due to bacteria which go through the walls of the gut at that point. In the majority of instances these adhesions cause no special harm and, unless they bind down a portion of the gut and bridge across, causing a stenosis, they had best be left alone. There are some individuals who seem to possess a lack of resisting ability on the part of the intestine to hold bacteria within the content of the gut. The majority of these individuals, however, have an infective condition of the tissues of the gut wall secondary to a long-standing chronic intestinal toxemia. There are others, who however great the toxemia, never get adhesions.

One not uncommonly sees cases where the entire ascending, transverse and descending colon have adhesions, essentially causing a long line of obstruction. Such cases are usually operative, requiring an ileosigmoidostomy or a cecosigmoidostomy for the purpose of short-circuiting the stenosed area. When, however, a diseased appendix can be eliminated in the case, it is best to consider the case as medical, because in the vast majority after an operative procedure, the adhesions re-form and very often more adhesions are added to those already present. I have ofttimes seen cases which have been made distinctly worse by operation for adhesions, and I am quite sure that I have also seen cases having adhesions in which the treatment for the underlying intestinal toxemia, finally caused the adhesions in the course of a few years to disappear.

Treatment.—In diagnosing intestinal adhesions the first consideration is to decide whether the symptoms in the case are secondary

PLATE XXI



Adhesion at left pelvic brim (sigmoid) due to long-standing saccharo-butyric toxemia. Barium by rectum. (X-ray by author.)

to a stenosis or not. If a normal, or reasonably normal transit through the colon is present, it is best to consider the case medical. If, however, distinct stenosis is present, the case is surgical. The medical handling of these individuals is along the lines of the underlying intestinal toxemia, which has already been mentioned, to which may be added the importance of massage and all sorts of physical means to improve the general condition of the abdomen. The one best thing for the treatment of adhesions that I know of, is carefully conducted abdominal massage, or vibration. No cases, however, improve distinctly unless the underlying intestinal condition has been handled wisely. The organisms which seem most responsible for the production of pericolonic adhesions are those of the anaërobic group—namely, the ones that are not distinctly pyogenic. One might compare the whole class of these organisms to the gonococcus which, as is well known, is capable of producing adhesions in the pelvis of the female without any pus unless it is locked up, as in the Fallopian tubes. But where they have gained entrance into the peritoneal surface, only a plastic type of peritonitis with adhesion results takes place.

INTESTINAL ACIDOSIS.

During the past years much interest and progress has been made in the study of the regulation of the body fluids as regards acidity and alkalinity, and in the development of our conception of the condition known as acidosis.

A number of conditions and diseases may disturb the reaction of the body fluids as regards acidity and alkalinity, among which may be mentioned anesthesia, diabetes mellitus, cyclic vomiting, gastro-intestinal conditions in children, pregnancy, kidney insufficiency, particularly that due to cystic degeneration of the kidney, post operative acidosis, severe diarrheas such as occur in entero-colonic conditions, nephritis, eclampsia, hemotoxic immune serums, etc.

Acidosis is unfortunately named because the condition is due to certain changes in the chemical processes of a body which normally is faintly alkaline, in which there is a tendency to reverse the condition by a diminution of the alkalinity of the juices bringing on the condition known as acidosis, but primarily the disturbance is a diminution of the alkalinity. The disturbance need not necessarily lead to a reduction of the alkalinity of the fluid of the body—the blood, for example—in fact there may be no alteration in the reaction of the tissues. The normal reaction in the body is maintained chiefly by these means. The first is the presence in the blood and

lymph of salts, chiefly the sodium salts, of two very weak acids—carbon dioxide and phosphoric acid. Of the two acids the former is the more abundant and important. In the blood and lymph, sodium carbonate (alkaline) and sodium dioxide (acid) are both present in such proportions as to give a nearly neutral reaction. If to this almost neutral fluid a stronger acid such as oxybutyric, lactic or hydrochloric acid be added there occurs an interchange, in which for every unit of the strong acid introduced into the blood there is liberated a unit of weak acid which possesses much less power of altering the reaction of the blood. The capacity of a free acid in a given concentration to alter the reaction of a fluid is dependent upon its ionization.

Therefore the constancy of the reaction of the blood is dependent upon a considerable amount of sodium carbonate with carbon dioxide and of sodium phosphate. When acids are added to the blood they combine with this sodium and liberated carbonic and phosphoric acid, both having very low coefficients of dissociation, hence producing a minimal change in the hydrogen ion concentration of blood. This factor alone is important in diminishing the changes in the reaction of the blood and body fluids when subjected to the addition of acid or alkali. In such conditions there is a rapid elimination from the body of these weak acids, phosphoric acid being eliminated by the kidneys and the carbonic acids by the lungs. To these weak salts constituted with the weak acids Henderson[2] has given the name "buffer substances." As a result of their presence in the body fluids and of the rapid elimination by the lungs and kidneys of the carbonic acid and phosphoric acid liberated from them, the blood and body fluids always preserve that constancy of reaction essential to life. When, however, a reduction in the so-called buffer substances of the blood has occurred, the condition known as "acidosis" is produced. The primary effect of such reduction is a diminution in the capacity of the blood to transport acids or alkalies. The acid most abundantly produced is carbonic acid, and when there is a reduction in the buffer substances, there is a reduction in the blood's capacity to carry carbonic acid. If this be marked enough there occurs an accumulation of carbonic acid in the tissues, generally with stimulation in the respiratory center. A more thorough ventilation of the lungs results with an increased removal of CO_2 from the tissues, which gives to us one clinical symptom of acidosis, namely, hyperpenia, and makes possible, in the estimation of the concentration of the CO_2 in the alveolar air the making of the diagnosis of acidosis.

An over-production of acid bodies is suggested by excretion in the urine of an excess of acid radicals, acetone alone in the milder cases and diacetic acid in addition in the more severe types, and these two with oxybutyric acid as the chief one in the most severe cases. The examination of the urine for these bodies is comparatively simple and need not be gone into here.

Since many cases of acidosis do not present large enough quantities of acid radicals in the urine, it is important to examine by a more direct means, namely, the alveolar air. To observe the content of CO_2 of the alveolar air, a means which is comparatively simple is the so-called Frizericia method. To determine the reserve alkalinity of the blood, either directly or indirectly, the method devised by Van Slyke[3] which estimates the CO_2 combining power of the plasma may be employed. A simpler and less accurate apparatus is that devised by Marriott, which estimates the CO_2 tension. It is accurate enough for clinical purposes in distinct cases.

With the exception of enterocolitic conditions in children, the cases of acidosis in connection with gastro-intestinal work are few. Here and there one meets with individuals, generally cases of the saccharo-butyric type of toxemia, in which there is more or less acidosis, and in fact, sometimes the typical symptoms such as the respiratory ones, a characteristic sweet odor to the breath, headaches, bradycardia, nervous excitation, vomiting, slight dryness of the tongue, a tendency to doze, dry skin, are present. In the most severe cases of acidosis convulsions are characteristic, and I have seen two such in chronic toxemias.

Not very much is known of how and why certain enterocolitic conditions cause degrees of acidosis. It has not been definitely proved that acidosis can be caused by these conditions, for usually when acidosis is distinctly present, we are dealing with a condition of disease of the kidneys, diabetes mellitus or some one of the conditions that have a more direct causative bearing. We do know that in children and young adults, and occasionally in older women, wherein it is not possible to diagnose anything other than an enterocolitic condition, acidosis can exist and is sometimes of rather grave importance. It seems in some way to be bound up with the digestion or metabolism of fat which upsets the reaction equilibrium of the tissues. It is present sometimes in late cases of cancer of the gastro-intestinal canal, and other fatal conditions in which probably the acidosis is caused by a condition of dissolution. No diagnosis of acidosis should ever be made without positive alveolar air findings.

Treatment.—Where no apparent cause for the acidosis exists, one's attention should be directed to an enterocolitic condition as being the cause, and careful examination of stool and urine is called for, placing the patient on a minimum diet, usually with the absence of fats, all the vegetables being boiled. After eliminating measures such as have to do with purgation and attention to the skin, comes the use of alkalies, preferably bicarbonate of soda in large quantities taken by mouth or rectum. Where a condition of saccharo-butyric fermentation exists this should be handled along the lines mentioned in connection with that subject.

In the last few years several cases of acidosis of the grave types (all of them in children) have been treated by blood transfusion with striking results. How this procedure favorably turns the course of a steadily downward case is difficult to understand, but that it may and often does I am firmly convinced. The blood of each donor was adult and none of the children were over nine years of age. Of course much smaller quantities than used in adults is sufficient, and the indirect method answers the best for clinical work.

References.

1 Kellogg: Medical Record, June 21, 1913.

2 Henderson: Clinical Studies on Acid Base Equilibrium and the Nature of Acidosis, Arch. Int. Med., 1913, xii, 153.

3 Van Slyke, Austin Jonas: Am. Jour. Med. Sc., January, 1917.

CHAPTER IX.

Catarrhal Conditions.

ACUTE DUODENITIS.

It is most probable that acute duodenitis is common enough, but it is rarely diagnosed. It is presumable that many instances of gastritis have instances of somewhat the same pathology in the duodenum. Acute duodenitis, however, rarely gives any symptoms as such and it probably is not an entity by itself, but rather an extension of an irritation or inflammation from the pyloric end of the stomach. When present there is no special treatment other than that for the causative condition, namely, the condition at the source of origin.

CHRONIC DUODENAL CATARRH.

While acute duodenitis, particularly that classified as the gastroduodenitis, can frequently be diagnosed by means of the duodenal tube, the diagnosis of chronic duodenitis is extremely difficult. Boas[1] hesitated to make a differentiation between localizing catarrhal processes in different portions of the intestine, although he was quite willing to do so between catarrh of the small and the large bowel. He stated that the presence of mucus in the feces, its shape (large or minute pieces) and color (whitish or bile stained), permits of the diagnosis of intestinal catarrh and its localization in the small or the large intestine, but the further subdivision of this rather long organ was not of practical value.

A few instances of definite chronic duodenal catarrh have been found at autopsy. Gaultier[2] mentions a patient where autopsy showed a definite chronic duodenal catarrh. Thus, I believe with Einhorn[3] that chronic duodenal catarrh is an entity and is possible of being diagnosed. I have met with several such cases in my own experience, where it was not possible to demonstrate a catarrhal condition of the stomach, but judging from the findings of the duodenal juice obtained by a duodenal tube the condition was perfectly manifest. The cases presented instances of distress two or three hours after meals with considerable flatulency, and two had diarrhea. There is usually some loss in weight, although this is not marked.

The diagnosis is made by noting essentially a normal test-meal and on aspiration of the duodenum a grayish-turbid fluid with thick particles of mucus and an alkaline reaction. The mucus normally obtained from the duodenum is colorless and very stringy, while that in cases of duodenal catarrh is different in color, and on microscopic examination distinct particles of mucus are visible. It is upon the presence of negative mucus in the gastric content and the flakes of mucus in the duodenal content that diagnosis is possible.

Treatment.—The treatment for the condition is essentially that for chronic gastritis in which hydrastis may be given immediately before meals in a mixture containing bismuth subcarbonate. The occasional employment of Carlsbad salts diluted with large quantities of warm water twice a day on an empty stomach; the patient lying on the right side afterward serves to good purpose.

ADHESIVE DUODENITIS.

The unfoldings of the operating room and the post-mortem chamber, lead one to believe that such a condition as adhesive duodenitis is a clinical entity. Commonly, adhesions are met with, not springing from the gall-bladder or elsewhere, but entirely associated with the duodenum. These are often inflammatory in origin and must have been caused by some inflammatory condition of the duodenum extending through the walls and involving the peritoneal surface.

It is generally considered that the duodenum is sterile of bacteria and that it is a part of the gastro-intestinal canal in which, when inflamed, the condition usually subsides. Whether these adhesive conditions are subsequent to a status of acute or chronic inflammation is not possible of demonstration. In several instances of sections of fresh duodenal walls that I have made there were distinct evidences of infiltration, thickening and more or less so-called catarrhal change in the epithelium. I believe that there are inflammatory conditions which can involve the pyloric end of the stomach, causing essentially a pyloritis with even large inflammatory masses, this condition extending out into the duodenum, and that many times in those instances adhesions are formed, gastric or duodenal in location. As to the running condition causing these adhesions and their diagnosis and treatment practically nothing is known. They apparently are symptomless or their symptomatology is so indistinct, or perhaps bound up with colloquial symptoms of a gastric disturbance, that it is not possible to diagnosticate such a condition, and generally, even when adhesions are present, it is not possible of being diagnosed

excepting when these adhesions so deform the duodenal cap that they are demonstrable by the X-ray. On the other hand, it is not uncommon to see instances in which the entire abdomen is negative but where adhesions of the duodenum exist, these being responsible for the condition; because usually when they are relieved the symptoms disappear. I have had several cases where adhesions bound down the junction of the first and second part of the duodenum, sometimes causing quite an angle, in which after a simple operation the entire symptoms disappeared. Therefore the possibility of this condition should be kept in mind, even though it represents one of the most difficult abdominal diagnoses to make, but by very careful X-ray work it can be done.

It is most probable that the cause of these adhesions is bacteria entering the duodenum surface either by way of the stomach or by ascending infection through the small intestine. While in health it is rarely possible to demonstrate the presence of bacteria in duodenal juice, in clinical work they are common findings. The instances are numerous of recovering B. coli from duodenums and it is logical to understand that these could migrate through the walls and cause an inflammatory condition with an adhesive peritonitis, no symptoms being present until the adhesions have existed for some time. The diagnosis of the condition is entirely a Roentgenographical matter, and the treatment in persisting symptoms is surgical.

SIMPLE CATARRHAL JAUNDICE.
(Gastro-duodenitis.)

This condition is due to a blocking from more or less inflammation and swelling of the common bile duct. While the duct alone may be involved, it usually is secondary to inflammation of the duodenum, this irritation and inflammation itself being secondary to a simple gastritis. The cause is usually a consequence of some gastric disturbance or the ingestion of deteriorated toxin-bearing foods, to which alcohol, rich spices, sauces and irritant poisons may be added. Sometimes it recurs in people because of an exposure such as chilling of the abdomen or sudden changes in temperature, insufficiency of clothing, mainly that about the abdomen. Most often, it is met but once in the history of a case.

A true epidemic form of this disease has been termed Weil's disease which has been accredited to a spirochete known as Spirochæta icterohemorrhagiæ. This condition is ushered in by a high fever, lasting one to two weeks, with a gradual decline in the second week, and is attended with considerable prostration. Albumin is commonly

found in the urine, and the spleen may be enlarged. Whether the simple catarrhal jaundice so frequently seen is a sporadic form of such an infection has not been determined, but probably Weil's disease is an entity by itself.

Simple catarrhal jaundice generally develops insidiously, rarely shows any increase in temperature, and if so it is very low. There is considerable prostration, slow pulse, an entire loss of appetite, some nausea, often vomiting, and there may be constipation or diarrhea due to the acholic condition of the intestinal content. There may be headache or a dull feeling in the head, the tongue is usually coated either brown or yellow, the breath is heavy and there is a bad taste. Shortly bile appears in the urine at which time there may be traces of albumin. Jaundice may be present early, although often it takes several days before it is manifest. The stools soon become clay-colored, the skin sufficiently dry and irritable to cause itching, the perspiration stains the clothing; depression is usually present, inability to do mental work; but generally there is no abdominal pain. The jaundice usually lasts from two to four weeks. If it lasts longer than four weeks it is wise to suspect that some other cause for the jaundice than simple inflammation is present, and in this connection malignant disease comes prominently forward in the majority of instances—I mean those in which pain is not present, because such are liable to be due to gall-stone conditions.

Treatment.—The more the patient is at rest the quicker the jaundice will subside, and for this reason it is best that he keep in bed. An initial dose of calomel is preferable to castor oil, this being followed by a saline. A local sedative like bismuth subcarbonate or cerium oxalate may be given every three or four hours during the day, these best being combined with an alkali such as sodium bicarbonate. The bowels should be moved by simple means such as sodium phosphate, and if there is much epigastric tenderness and soreness, the drinking of hot water or hot applications is advisable. The patient should have abundant water to drink, and in the presence of nausea, milk of magnesia in teaspoon doses in hot water every three or four hours is worthy of a trial.

It is best to begin the treatment by purgation and starving, allowing nothing but water for one or two days. Nourishment should be begun in the form of bouillon or hot broths, tea, toast, thin oatmeal gruel, or some other thin gruel. Malted milk may be given, and in a day or two eggs if they are well tolerated. As a rule milk is not a good food for this condition, and the same may be said of fats of all

kinds. It is best that no liquid be given cold, and the patient should be kept essentially on a soft diet until the jaundice has disappeared.

To relieve internal congestion and to increase the secretion of the skin and stop itching, a daily hot bath is advisable. It is of service especially in removing the irritating crystals that occur on certain parts of the body from the perspiration in this condition. The itching also may be controlled by a lotion of 5 per cent. carbolic acid in alcohol. Sometimes a moderately strong solution of sodium carbonate or a saturated solution of sodium bicarbonate serves to good purpose.

As the patient improves the diet can be enlarged, but it is best to withhold the cellulose bearing vegetables until late. Fruit may then be tried but it is best to withhold fats until the stools have taken on a distinctly yellowish tinge. If the jaundice lasts more than ten days or two weeks, ammonium chloride in ½-gram doses, three times a day, after meals, may be given. Ammonium chloride is supposed to increase the secretion of the mucous membranes, and in that way deplete the duodenum of its inflammation. Probably it also increases the secretion of the bile ducts as it does that of the bronchial tubes. The following prescription has been advised:

℞ Ammonium chloride ..... 10 Gm. or c.c.
Syrup of citric acid ..... 25 Gm. or c.c.
Water, up to ..... 100 Gm. or c.c.
Mix and label: A teaspoonful, in water, three times a day after meals.

DUODENAL DILATATION.
(Kinking of the duodenum.)

Of late dilatation of the duodenum has awakened considerable interest. Albrecht[4] pointed out that this segment of the duodenum has, under normal conditions, not a round contour but a distinctly flattened circumference; and Codman,[5] a few years ago emphasized this fact and presented casts of a number of duodenums showing definite evidence of compression. These observations were supposed to be of interest in connection with the fact that the terminal portion of the duodenum passes behind the root of the mesentery and lies on the vertebral column and aorta, and that in the erect posture of the human being this transverse part of the duodenum is more or less compressed by the mesentery and its contained superior mesenteric artery.

In numerous text-books and articles this fact of the course of the duodenum in connection with the mesentery and superior mesenteric

artery has been mentioned as a prominent factor in connection with dilatation of the duodenum, particularly in ptosis cases. After an observation of many thousands of cases of ptosis, so far as Roentgenographic examination and demonstration can go, I do not believe that this is a practical fact in connection with the cause of dilatation of the duodenum. It is true that one occasionally sees a largely dilated duodenum in connection with ptosis, but too often in the worst cases of ptosis it is not present, and I state this even in the face of the fact that traction on the mesentery generally causes more or less complete inclusion of the duodenum. It is far more probable that this condition is due to other factors than the one mentioned. Easily the most common cause of dilatation of the duodenum is a marked state of ileal stasis for whatever reason, not as Jordan[6] suggests because this is due to sagging of the small intestine and a pulling on the mesentery, but to a backing up that occurs in the intestine plus the onward running peristalses of the stomach causing a dilatation of the duodenum, it being the zone which would receive the greatest amount of to and fro pressure effect. The next most common cause is an extension of a gastric atony outward through the pyloric muscle and involving the first part of the duodenum, perhaps the second and third parts being essentially normal in caliber, although the dilatation gradually subsides so that it is not possible to sharply delineate that the dilatation is only in the first part. This is commonly seen in ptosis cases, but in far more instances in cases of distinct gastric atony with a relaxed pylorus, the condition being due to a long-standing intestinal toxemia with effect upon the sympathetic plexuses in the rear of the abdomen, in that way interfering with the musculature tone of the stomach and first part of the duodenum which are essentially the same organ.

Symptoms.—The symptoms of chronic dilatation of the duodenum are those of an infrapapillary constriction—that is, obstruction below the entrance of the common bile and the pancreatic ducts into this portion of the bowel. Or, on the other hand, they may be absolutely negative even in the presence of the most extensive dilatation. I have seen duodenums almost as wide as the pyloric end of the stomach with no symptoms such as are described below.

It is believed that the persistently occurring vomiting, in most instances the vomitus containing bile, is distinctive of this condition. It should be remembered here that such may be present in migraine and in many gastric disturbances in which bile is found at the end of gastric digestion.

Pain in the upper part of the abdomen is generally referred to the right hypochondria, as a rule described as an aching, but it may be severe so as to suggest biliary colic of a slight degree. In my opinion when any symptoms due to the dilatation are present, it is never a pain—simply an uncomfortable feeling which is so closely associated with other conditions that it is not possible to identify it as due to a dilated duodenum. Other symptoms mentioned are those connected with ptosis, obstinate constipation and vague toxic symptoms such as headache, hypersensitiveness, unstable nervous function, etc. In my opinion none of these are characteristic of chronic dilatation of the duodenum.

It has been suggested also that the clinical picture of some cases is very suggestive of cholecystitis. Vanderhoff,[7] I feel, rather overstated the danger of mistake in not being careful enough in getting the detail of a good history, because the symptomatology of gall-bladder pathology is usually so distinctive that a careful clinician rarely meets with this confusion. Still, it should be mentioned that occasionally the abdominal distress may occur over a period of months and there may be some tenderness on pressure so closely associated with the gall-bladder region that differentiation will be difficult. The same may be said between duodenal dilatation and peptic ulcer.

An accurate diagnosis can only be made by a competent Roentgenographical examination, when the condition becomes readily manifest.

Treatment.—The treatment of chronic dilatation of the duodenum is usually medical and rarely surgical. In the presence of ptosis, not so much because of the arterio-mesenteric theory of the cause, but because these patients have distinct conditions of atony and general deficiency, the ptosis requires full consideration or no benefit will be achieved. This means all that has been mentioned in connection with the treatment of gastroptosis in the volume "Diseases of the Stomach," or that dealing with enteroptosis later on in this volume. If vomiting is present with accumulation of bile in the stomach, lavage may be practised. By far the largest number of cases, however, have a chronic intestinal toxemia which requires attention, and unless this is handled with success benefit in the dilated duodenum is not met with.

Occasionally one meets with cases where there has been a long history of starvation and perhaps some pseudo-clinical symptoms of acidosis. In these it might be judged wisest to resort to an operation, of which several different forms have been mentioned—one, widening the duodenal slit in the mesentery; another, duodenoje-

junostomy; another, resection of the right half of the colon by ileocolostomy, and some even have resorted to the operation of gastroenterostomy, which I think is unwarranted in this condition.

In my opinion, careful medical treatment directed to the ptosis, the intestinal toxemia, and the general condition of the patient, this being carried out over lengths of time, supported perhaps by large doses of nux vomica and constructive tonics, abdominal support, diet, etc., would mean that practically no cases of dilatation of the duodenum would require operation for the condition.

ATRESIA OF THE DUODENUM.

As is well known, atresia of the duodenum is not such an uncommon congenital condition. While the number of recorded cases is by no means large, many have not been reported and have passed unrecognized in the absence of post-mortem examinations.

Little and Helmholz[8] were able to collect, including their own, only twenty-seven cases of atresia of the duodenum above the papilla of Vater, Silberman reported twenty-four cases of atresia of the duodenum; Cordes,[9] forty-eight cases of atresia and nine of stenosis of the duodenum.

Unless great care is taken the possibility of such a condition is not thought of until the treatment is rather hopeless, and most often is missed entirely. Most of the cases are classed as malnutrition, spasmodic stenosis, or hypertrophic stenosis of the pylorus.

The characteristic symptoms are persistent and characteristic vomiting, constipation and change in the character of the stools, distention of the abdomen, visible peristalsis, anuria, emaciation, jaundice, other congenital malformations or hydramnios in the mother. The vomitus is usually collective having no necessary relation to feeding. The bile will be present or absent as the obstruction is below or above the papilla of Vater. A stomach tube passed in suspected cases would demonstrate the size of the stomach. If the stomach is greatly dilated the abdomen will be prominently distended and splashing sounds may be elicited. The anuria is due to the fact that no fluid is absorbed from the stomach or sufficient amount passed into the intestine.

The diagnosis lies between a spasm of the pylorus, congenital hypertrophic stenosis and congenital atresia with complete obstruction. Spasm of the pylorus may be present shortly after birth, but often does not cause symptoms for days, weeks, or even months. The same may be said of hypertrophic pyloric stenosis. It is char-

acteristic of the spasm in the hypertrophic cases that in these the emaciation and malnutrition are slower in their development than in atresia of the duodenum.

The treatment is surgical—usually a quick gastroenterostomy. The prognosis because of the tender age of the patient is bad.

BENIGN STENOSIS OF THE DUODENUM.

Benign stenosis of the duodenum represents about the same causes as the benign stenoses of the pylorus. These then may be classified as tumors which may cause a narrowing of the lumen of the pylorus and duodenum from without, such as those of the pancreas, gall-bladder, etc.; adhesions around the pylorus or duodenum which may occasionally interfere with its function, and may lead to kinking and distortion of the entire lumen; and angulation or kinking of the normal duodenum at the peritoneal point of fixation, at the pars superior duodeni.

Depending upon the degree of stenosis these cause the well-known symptoms of pyloric obstruction—namely, the colloquial symptoms of gastric distress, nausea at the end of the day which requires vomiting for relief, and this may be present when large amounts of fluid are in the stomach, or, stagnant vomiting—that is the vomiting of stagnant material, usually material which is left over in the stomach some hours after ingestion. More or less emaciation is usually present and commonly visible peristalsis in the gastric region with distention of the stomach which may be in such an active tone in an effort to empty itself that one can actually feel the organ through the abdominal wall. Usually, smaller than normal quantities of urine are passed by these individuals because of the limited amount of resorption of fluid from the stomach, and there being little that is gotten out into the intestinal canal where resorption is most active.

The test-meals are characteristic of stagnation, fetid from the presence of sulphurated hydrogen gas, darker in color than normal and in larger quantity. Usually there is a higher acidity because of the concentration of hydrochloric acid, and commonly the X-ray examination shows the characteristic retention after six hours and the globular shape of the organ.

Often in these cases the symptoms of gastric stagnation are not distinct and one requires considerable examination to diagnosticate the presence of stenosis. When tumors are present which narrow the lumen, it depends upon whether these are fixed or movable, and not uncommonly patients learn which way to sit and lie so as to have the

weight of the tumor fall away from the viscus and in this way relieve the symptoms of distress. Adhesions about the pylorus and duodenum, however, are usually fixed and the symptoms here are usually those simulating somewhat cases of ulcers, cholecystitis, even perhaps duodenal dilatation. In the presence of angulation at the fixed point of the duodenum this can very easily be diagnosed by trying to pass the duodenal tube which contains bismuth, and observing a sharp kinking of the tube at the left margin of the spinal column. It must be manifest, therefore, that the diagnosis of this condition is largely a Roentgenographical matter, because pressure effects upon the hollow viscus or the tugging, pulling or binding of adhesions cannot be diagnosed in any other way.

Treatment.—This depends upon the degree of obstruction and its character. In the presence of tumors, operation is in order; the same may be said of adhesions which bind down this portion of the gastro-intestinal canal. In sharp angulation, however, the treatment may be conservative and usually is successful if it is carried out thoroughly. It is essentially that of the treatment for ptosis, which in my opinion, is the cause of sharp angulation at the fixed duodenal point.

DUODENAL ULCER.

The author believes that duodenal ulcer should be considered as peptic ulcer and all that pertains to it will be found in connection with my work on the stomach, in which duodenal ulcer is fully treated.

ACUTE ENTERITIS.
(Enterocolitis.)

This term represents an acute catarrhal inflammation of the mucous membrane of the small intestine, as well as the upper portion of the large bowel. The terms acute inflammatory diarrhea, enterocolitis and ileocolitis are synonymous. As a rule it is not possible to differentiate this condition sharply from acute dyspeptic diarrhea due to the eating of indigestible foods at certain times in the year, notably in the summer months. Nor is it possible always to differentiate it from certain infective diseases with increased intestinal manifestations. It must be remembered that certain specific infective diseases such as typhoid fever, cholera, dysentery, etc., are not represented in this classification.

Some individuals have a susceptibility to acute enteric conditions manifested most often in warm weather and under conditions of contamination of foods, usually those of fruits or heavy cullulose-

bearing carbohydrates. The condition is not uncommon in children following a chilling of the surface of the body during the hot days. It is also found in certain cases of mineral poisoning, poisoning by toad-stools or other noxious substances.

The mucous membrane of the bowel is involved with almost equal severity from the stomach to the upper portion of the large intestine. It usually is swollen and reddened with the crust of the valvulæ conniventes presenting an inflammatory appearance. The surface is covered with mucus which may be blood-tinged, and the lymphatic follicles as well as those in the patches may be swollen, some occasionally showing small round ulceration.

The same form of this condition may be met with in cases of chronic nephritis or uremia, and is commonly found in cirrhosis of the liver and other conditions producing portal obstruction, or in association with malignant disease of the bowel, or other lesions.

Symptoms.—The clinical manifestation of enteritis is that of the acute dyspeptic diarrhea, the movements being more or less offensive, according to the degree of putrefactive change and usually contains a small amount of visible mucus. When the large intestine is principally affected the amount of mucus may be considerable, but in most cases the discharge is thin, watery and brownish. Sometimes the symptoms are very marked. In some cases of intense and localized enteritis, paresis of the affected segment of the bowel may occasion intense constipation or even complete obstruction of the bowel.

The patient usually suffers with colicky pain and more or less abdominal soreness from the beginning. Distention of the abdomen takes place and loud rumbling or gurgling sounds are frequently heard. When the lower part of the ileum is involved the local symptoms may slightly suggest appendicitis. In such cases rigidity of the abdominal muscles in the area of pain and tenderness, the occurrence of fever, or vomiting and leucocytosis may still further confuse the diagnosis. Development of the diarrhea is usually the determining symptom suggesting enteritis. In the beginning of the attack nausea and perhaps vomiting may be present, the tongue may become coated and dry, the appetite wanting and there may be more or less distress after eating. When the upper part of the small bowel is especially involved active gastric symptoms may be a conspicuous feature in the clinical manifestations. Fever, more or less high, is usually present. During the time that the diarrhea is on there is more or less loss of strength and general debility. Sometimes considerable symptoms of shock such as the surface of body becoming cold and moist and coldness of the extremities, etc., may be present.

What has been termed cholera nostras or cholera morbus is an intense form of this condition, having about the same relation toward many cases of enteritis of adults as cholera infantum bears toward enterocolitis in childhood. Such intense conditions usually follow the eating of fish, shellfish, unripe or tainted fruit, the drinking of large quantities of fermented liquor, beer, or a chilling from the outside from exposure during hot days. The symptoms are those above mentioned, being of the marked type.

Diagnosis.—The recognition of enteritis as a rule offers no great difficulty. The history, onset of abdominal pain of a colicky nature and the diarrhea are characteristic. In some cases considerable difficulty may be met with in distinguishing enteritis from appendicitis. In appendicitis it must be remembered that the local symptoms are more persistent, nausea and vomiting, and perhaps chills are a distinct feature with the increasing hyperleucocytosis. Obstinate constipation, not necessarily diagnostic, is strongly suggestive of appendicitis.

The prognosis as a rule is favorable. In the old or enfeebled, however, or in children, the disease may be one of considerable gravity. Sometimes the condition ends in a subacute or even a chronic intestinal disturbance.

Treatment.—Prophylactic treatment is of considerable importance. It has to do with preventive measures, the taking of irritative foods and the like, especially in the hot season and under conditions of confinement such as in camps, barracks, etc. Individuals should be careful of getting the surface of the body chilled during summer weather, particularly in tropical countries.

The treatment of the condition is that of an ordinary diarrhea, the patient being kept strictly at rest until all of the symptoms have subsided. Warm applications to the abdomen not only afford relief but have a controlling effect upon the peristalsis.

As soon as the nature of the disease is recognized the first indication is to remove the irritating substances from the intestinal canal. A full dose of castor oil or the use of calomel followed by saline may be employed, although the best practice is to wash out the canal by means of trans-intestinal lavage containing about 9 grams each of sodium sulphate and sodium chloride in a liter of water.

The diet should be restricted to fluids such as boiled milk, broths, gruels, albumen water and the like. When gastric symptoms are present it is wisest to withhold food for some time. Bismuth salts, mixed with lime water, etc., may be used, astringents and opiates rarely being necessary. Sometimes an initial injection of morphine is grateful, particularly when the symptoms are acute and the general

system is showing evidence of collapse. In the latter instance hot applications over the abdomen are advisable together with hot drinks and the addition of small doses of brandy or other stimulant. In the subsidence of the condition care should be taken that the solid diet is not resumed too quickly or the patient gets about too early. Usually in the course of several days the condition is under control and additions to the diet can be made.

CHRONIC ENTERITIS.

Chronic enteritis is a catarrhal condition of the small intestine and upper part of the large, usually without ulceration, and commonly a sequel to acute attacks. The disease usually takes place after repeated slight attacks of acute enteritis, and it is commonly found in other conditions of the bowels such as carcinoma, intestinal obstruction, fecal infections, etc.

Pathology.—The intestines usually present evidence of long-continued inflammation of the mucous membrane. The surface is covered with extensive secretion, mucus or muco-purulent. There are evidences of the thickening of the mucosa or of erosion and atrophy. Such conditions, even without ulceration, in time may heal with cicatrices; especially is this true in the large bowel. Not uncommonly polypus formations are found surrounding ulcers or independent of any ulceration, the polyporic formations being generally secondary to the ulceration of the mucous membrane.

Symptoms.—The characteristic symptom of chronic enteritis is persistent or intermittent diarrhea. In the intermittent type of case the movements are variable, sometimes normal and at other times soft, pultaceous and foamy, and at other times very fluid. In some cases of chronic enteritis there is present a persistent constipation, in which instance the stools are light colored or gray firm masses coated with mucus. When the involvement is mostly in the large intestine mucus is a pronounced feature.

The abdomen is usually distended although the examination may be negative. Usually the individuals persist with this condition without much impairment of health. Some, however, show emaciation, general depression, and a variety of nervous symptoms.

The diagnosis is usually difficult to distinguish between chronic enteritis and the functional disturbances of the intestine on one hand and mucus colitis and certain primary nervous disorders on the other. It is important to remember what Nothnagle stated, that in a long experience he had never met with a normal person with normal de-

fecation that had either macroscopic or microscopic traces of mucus. While this is true it must be remembered that at times one meets with mucus in the stools in instances of people in which the condition is entirely due to a nervous disorder, such as in some cases of so-called mucus colic. Then there are instances of nervous diarrhea which are extremely erratic in different individuals but pursue a fairly regular course in the same individual. For instance, one person is aroused early in the morning by a desire for defecation and three or four loose stools follow without pain, and these cease until the same hour the next morning when they are repeated. Others are interrupted in the midst of a meal, usually the midday one, by two or three loose movements, and then there is no recurrence of these until the next day at the same time. The examination of stools of such individuals generally fails to show any evidence of impaired digestion in the food products, nor is there any mucus present. Such individuals often show definite reaction in the disturbed reflexes and belong mainly to the "nervous" class. Not uncommonly one meets with a syphilitic or a tubercular person who has this tendency to looseness of the bowels in which the diagnosis may be somewhat difficult and only can be determined by the clinical course and results of treatment. The urine should be gone over for the purpose of establishing a diagnosis of chronic intestinal toxemia. It is not uncommon to find in cases which might be classified as chronic enteritis a condition in which there is putrefaction existing mainly in the small intestine. These persons, in my experience, recruit most of the cases of so-called chronic enteritis, the treatment of which is one of handling it from a toxemia standpoint as well as treating it as a catarrhal condition. Subject matter pertaining to this will be found in Chapter I.

Treatment.—The care of these conditions is usually a matter of weeks, months or years. The prognosis is generally uncertain but persistent treatment is usually effective.

The first indication is to observe carefully for the presence of any focal infection existing about the sinuses in the head, teeth, fauces, etc. Such not being present, a careful examination of the stools and urine for the purpose of noting whether a chronic intestinal toxemia exists is next in order. As a rule there should be a modification in the habits of life, and avoidance of fatigue, exposure and similar causes.

When a condition of intestinal toxemia exists, depending upon the character, the treatment is for the condition found, with the following added suggestions:

As a rule these individuals are debilitated and require considerable tonic measures in which nux vomica answers the best purpose. This may be united with the organic forms of iron. Where symptoms of diarrhea exist, a dietary proper for such a condition is in order, and when one of constipation is present, an anti-constipation diet. Astringents such as acetate of lead, sulphate of copper, bismuth salts may be employed, although they serve no useful purpose so far as my experience goes. Intestinal antiseptics may sometimes be employed with benefit. When there is an involvement of both the small and large intestines, or an involvement of the small intestine alone, trans-intestinal lavage with about 9 grams of sodium sulphate and chloride of soda or bicarbonate of soda, perferably in camamile infusion is in order, these taking place every day or every other day, and kept up for several weeks. Not uncommonly the use of fluid extract of hamamelis or fluid extract of hydrastis may be employed, the first in a saline solution—about 1 to 125 parts; the second about 2 cubic centimeters to the liter of saline solution. The giving of opiates should never be employed.

COLITIS.

(Mucus colic, Muco-membranous or catarrhal colitis.)

Inflammation of the colon frequently occurs as an accompaniment of general catarrhal inflammation of the bowels or enteritis, and is a common accompaniment of organic disease of the large bowel such as carcinoma, stricture, fecal infection, and the like. It is met with also as a manifestation of specific infections such as dysentery, etc. However, it occurs as an independent disease, which is the form under consideration.

Until rather recently it has been debated whether chronic colitis is an entity. Some observers have claimed that inasmuch as it represents the inflammation of the wall of the colon and that at post-mortem no evidences of inflammation existed, that it was not an entity. But in more careful attention to the minute study of the large bowel this view has proven to be erroneous, and it is now known to be one of the commonest disorders of the human being. The thickening of the mucosa, excessive mucus formation, follicular inflammations, ulceration and so on are observed with ecchymotic spots here and there. Infiltration of the submucosa and even general thickening of the wall are not uncommon. I believe with Daniels[10] that the following classification is the simplest for understanding the condition:

1. Primary, idiopathic colitis (mucous, muco-membranous, or catarrhal).

2. Secondary or consecutive colitis: (*a*) bacterial in origin; (*b*) protozoan in origin; (*c*) unclassified origin.

A working classification is that based on the character of the discharge:

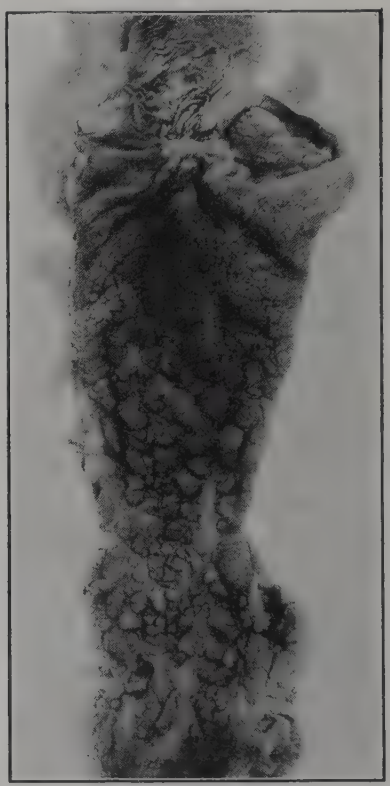


Fig. 63.—Chronic colitis. Chronic inflammation of colon with diffuse edema, thickening of all coats and chronic ulcerations. (*McMahon* and *Carman.*)

1. Colitis with mucus discharge.
2. Colitis with all other forms of discharge.

It is probable that the primary or idiopathic group are largely neurological in origin, although here dietetic reasons often enter in as ample causes. In the secondary or consecutive groups those bacterial in origin certainly represent by far the greatest number of all of the colitis cases that are seen. While it is not always possible to prove which are causal bacteria, and which are, so to speak, accessory after

PLATE XXII

Chronic colitis, dilatation of the catarrhal right colon, the whole viscus in irritation as shown by the excessive peristalses throughout. Fermentative type of toxemia present. (X-ray by author.)

the fact, *i.e.*, which exert an influence once a lesion is initiated, the fact remains that careful study of the stools of these individuals commonly prove that they have certain bacteriologies which correspond with certain pathologic changes.

We have on the one hand the bacteria of the B. coli group in which the pathology most often seen is that of an atonic type with a flattening and thinning of the mucous membrane, generally a dry surface and attenuation of the wall of the gut. In such instances the pathology mentioned in connection with long-standing intestinal

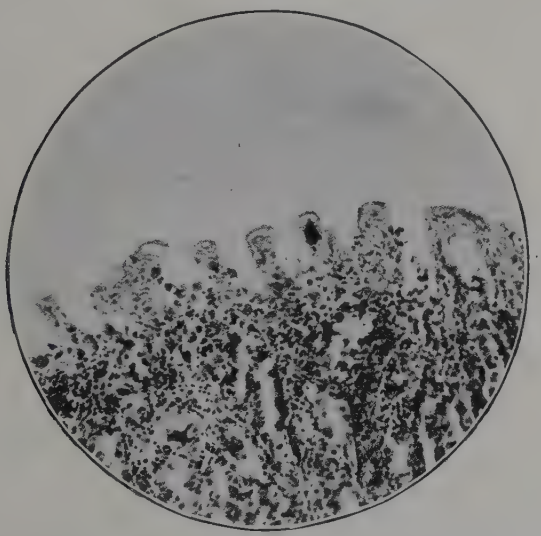


Fig. 64.—Microphotograph of mucosa from a case of chronic colitis (diphtheritic colitis). ×100.

toxemia will be noted. On the other hand are the cases, mostly of the saccharo-butyric type, in which there is less destructive effect upon the mucous membrane and walls of the gut, perhaps considerable hyperplasia being present. There is of course destruction in the glandular secreting apparatus, although the mucus elements usually predominate and the walls of the gut are wet. These cases are more particularly of the hypertrophic type of pathology and are represented in the fermentative class of intestinal toxemia, due to high anaërobic content of bacteria, most often the putrefactive anaërobes of the B. aërogenes capsulatus and Gram-positive coccal type.

The neurological cases which are additionally due to indiscretion in eating represent by far the majority of the remaining instances of chronic colitis. Leaving the definitely dietetic ones out of considera-

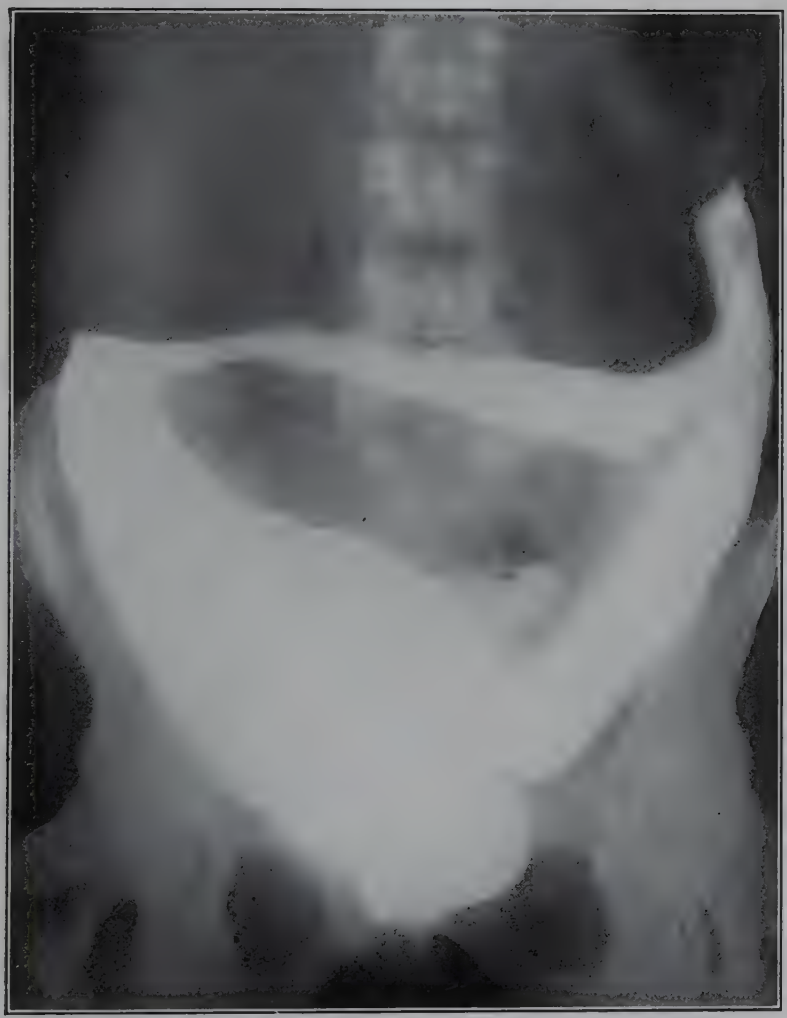
tion they are met with in individuals usually poorly nourished, thin, anemic, having low powers of endurance, sufferers from neurotic and psychasthenic states (commonly recurring from time to time), and those of a nervous, excitable and restless type, usually women, although not at all infrequently men. This neurological type is also observed in individuals who, upon examination disclose evidences, usually more or less combined, of disturbances of the endocrine glands. These disturbances may be manifested in the body make-up or in the skin reactions, or noted by the irregularity that exists in the balance between the vegetative and the autonomic systems, and by various facts connected with the sexual or intellectual spheres. These are the cases which have been described under various etiological combinations, the most common of which is that they are secretory and motor neuroses of the large intestine, with two types of pain—a more or less constant aching in the lower abdomen and a paradoxical colicky pain always relieved by defecation or an attack of mucous colic. Such cases are not clinical entities; they are usually a collection of symptoms associated with various organs in the study of the human being as a whole, and when a diagnosis of colitis is made, the treatment is suggested along the line of the findings in the individual case.

Commonly, too, these individuals have more or less kinking and partial obstruction of the colon from adhesions or pericolitis, a dilatation of the colon and visceroptosis, perhaps a chronic appendicitis, inflammation in the uterus or appendices, or post operative adhesions, etc., resulting from operations on the abdomen that had been met with. In this type of colitis, matters pertaining to diagnosis are sometimes very difficult. Often much can be accomplished by careful investigation of the stool and examinations of the urine, the abdomen, systematic sigmoidoscopic examination of the pelvic colon, etc.

Because there are so many complications connected with each type of case, each requires a minute study of the body as a whole. Unless this is done there occur the many short-comings that make the understanding and the treatment of the idiopathic type of colitis so difficult. It is for this complex reason that it is impossible to classify types of mucous colitis in any uniformity of nomenclature, in etiology and in symptomatology. Remember that this type of colitis is a disease of civilization, of inherited nervous instability, of ingestibility of many of our foodstuffs, occasionally, of uncertain anatomic and many other conditions, so that in a series of these people there is always a striking difference between any two of them as to the important causes and the bases of treatment.

PLATE XXIII

Marked general colitis with absence of haustral contractions, prolapse of the transverse colon and angulations at arrows. Indolic type of toxemia. (X-ray by author.)



Chronic colitis; 8 years' duration, small smooth colon, absence of haustra especially from hepatic flexure onward. Ileocecal valve incompetent. (*McMahon* and *Carman.*)

As far as the abdomen is concerned they have, like the bacterial cases, a tendency to sluggish movements of the bowels in the atonic cases, the stools perhaps being of large size, and the patient having to resort to laxatives and various means to sufficiently stimulate the muscles of the colon. Not uncommonly in such cases the use of cathartics will bring on more or less flatulency and colicky pains, the stools perhaps being covered with mucus from the glands of Lieberkühn. Other cases again present various spastic types of constipation with perhaps hypertonicity in the musculature, the individual having soft and mushy stool, with even a tendency to diarrhea. Not uncommonly there is a history of attacks of mucous colic which sometimes are so severe that they present a striking picture of acute disorder. These attacks occur most commonly when the individual has made more physical expenditure than the vitality warrants, and thus a nervous unbalance occurs causing an explosion in the abdomen which represents itself in intense pain, colic, tenderness on pressure and several movements of sometimes alarmingly large amounts of mucus, the whole condition subsiding in a few hours to several days. Always in such instances the general nervous system of the individual should be taken into consideration as the main etiological factor. The other cases, again, present more or less continuous neurasthenia, or history of excessive literary effort, worry about a wayward husband or child, some other domestic worry, or perhaps the initiation of a menstrual period. As I stated before, malnutrition is often a prominent feature in these individuals, not a few of whom I have seen subsisting on seven hundred to fifteen hundred calories of food a day when they should have had at least twice or three times as many. At times, too, they partake largely of coffee, alcoholic drinks, tobacco, and these add their toxic factors particularly manifest in an attack of nervous instability.

Visceroptosis and uterine displacements have been mentioned, and appendicitis and other focal infections may cause the individual to be more liable to these attacks.

Lastly should be recorded that a very large proportion of cases of chronic colitis have the colitis as a secondary condition from an intestinal toxemia. The mucous membrane here simply expresses a resulting condition, yet this resulting condition may bring on such pathology that it runs on as an entity. Heretofore these cases have been considered very difficult but in my opinion they are the simplest of all types of colitis to treat. The important question in handling colitis is to eliminate all causes such as are represented in the dietetic and neurological group, those with endocrine gland disturbances, etc.

Coming down to no sufficient cause, a careful examination and the bacteriological study of stool will ofttimes present an underlying toxemia from a bacterial infection which may be removable, or changed by diet. The subject then, both as to diagnosis and treatment, is bound up in the treatment of the infection which has already been presented. What follows merely represents the general treatment of colitis—not that secondary to an intestinal toxemia.

Symptoms.—The principal manifestations of colitis are diarrhea, local pain and tenderness. However, all of them may not be present. Usually, there is more or less abundance of mucus in the stools which may be slightly blood-tinged. In severe cases small mucus blood-tinged discharges, resembling those of simple dysentery may occur, and may be frequently repeated. In subacute cases fecal matter with mucus and blood may be passed. Abdominal pain, especially in the region of the sigmoid flexure or anywhere along the course of the colon is commonly present and sometimes there is extreme tenderness on pressure. The author has seen instances of fever and general prostration of more or less intensity.

Most often the pain is indefinite in degree, variable in situation, not relieved by suitable laxatives nor any natural movement of the bowels. It is invariably the pain due to ordinary constipation and colic, although in some cases it may last for several days. The pain is usually worse toward evening and after hot drinks. Pressure often makes the pain worse, which is unlike the relief generally experienced by pressure in ordinary colic. If the attacks appear periodically the pain gradually appears and tends to get worse up to a climax, then a furious diarrhea sets in bringing relief.

A history of habitual constipation is generally present. Not uncommonly one cannot get a history of excess of mucus from patients—many people not being observing in this way. With a history of constipation there may be attacks of looseness of the bowels, particularly when excessive amounts of mucus are present from irritation of the retained feces. Flatulency with distention is commonly observed, the flatulence and discomfort usually increasing at night and after hot drinks.

The colloquial symptoms of indigestion are commonly met with in these cases. It is my opinion that the commonest cause of hyperacidity of the stomach, intermittent achylia, gastric atony, as well as disturbances of sensation of the stomach, is represented in cases of chronic colitis. Ofttimes they are described under the above mentioned headings and treated by various gastric methods when the real cause of the condition is in the large intestine. As my exper-

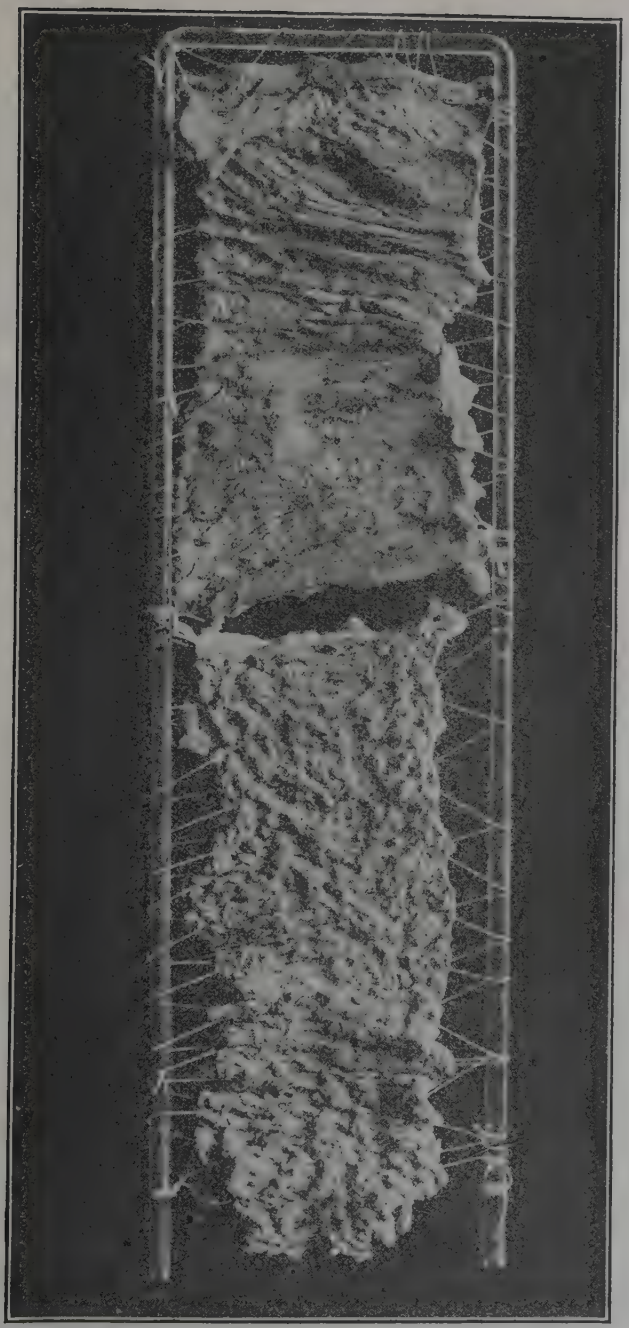


Fig. 65.—Colon of diphtheritic colitis, showing infiltrated hyperplasia and organization commingled with areas of atrophy of the mucous membrane. A late stage of chronic colitis having marked pathology due to long-standing B. coli infection of the gut tissues.

ience increases I think that the etiology of many gastric disorders is present in the intestinal canal and that much of the treatment of years gone by directed to the stomach itself has been false. This has been learned by me from the substantial results obtained in instances where the colon is responsible for the gastric disturbance and distress, and not the stomach for that in the colon. In such individuals there is commonly an absence of robustness and energy, often actual wasting and emaciation. Pallor and mild anemia are usual. With suitable stimulation these patients often exhibit quite marked energy and briskness but it is not long maintained and the reaction is liable to be severe.

In all instances a careful examination of the individual as a whole, taking into account the nervous reflexes, various evidences of endocrine organ disturbance, and all matters that pertain to sociologic and economic factors under which the individual lives, together with a careful examination of the stools bacteriologically are required. Relief in instances of chronic colitis is easy; cure is difficult. But it is rendered comparatively easy if individual work is done with individual patients. This is the one condition in medicine wherein generalization counts for very little. It is the non-observance of the latter fact that has made the cure of chronic colitis such a questionable matter in medicine. It is really a provision of the Lord that the course of chronic colitis is possible with fair health in the individual and that in instances of acute attacks the results from most any form of treatment are favorable. In my opinion, however, we must go beyond this point, and it is simple enough to do so if we make a careful, complete examination of the patient and take all factors into consideration.

The X-ray serves a useful purpose in these diagnoses. In the irritative form of toxemia (fermentations) increase of haustral contractions and perhaps marked spastic states may be noted. In fact spastic conditions of the colon are always due to this type of toxemia and consequent irritative colitis. In the indolic and mixed forms of toxemia absence of haustral contractions is commonly observed. This most often is in the left side, perhaps in the sigmoid region alone. Their absence, as the author has pointed out, is due to destructive conditions in the neurological tissue of the gut. (See Intestinal Toxemia.)

Treatment.—This resolves itself into the relief of the attack itself and that calculated to remove the causes of the disease. Rest and warm applications to the abdomen with careful regulation of the diet, administration of remedies to allay the intestinal irritation are the important features of treatment. A dose of castor oil followed before it has time to act by a saline soap enema is worthy of general utilization.

Of late it has been my custom to do a trans-intestinal lavage with a hypertonic saline solution getting a quick emptying of the gastro-enteric canal, administering bromides in sufficient doses with warm applications to the abdomen, no food for the first day, and then the employment of a soft diet, and I think my results have been better than with the older methods of treatment. Opiates should not be used, not even in the form of opium suppositories.

The treatment of the cause depends upon what the cause proves to be. This, as mentioned before, may be dietetic, neurological, disturbances in the endocrine organs, which are commonly inherited and should be recognized early in life for their proper treatment rather than the treatment of the end results such as we have been doing in the past, treatment of intestinal toxemia in cases wherein the colitis is secondary, surgical procedures for the removal of focal infections, appendicitis, etc., medical procedures for visceroptosis, and so on.

The dietetic treatment is best expressed in an anti-constipation diet, about as follows:

The plan of the diet is to eat three meals a day with nothing between times. The food should be of the normal kinds and simply cooked.

Drink at least five glasses of water a day, preferably before meals and between them, not during the meal. Any of the customary foods can be eaten but do not take more than two eggs in a day and meat, fish and fowl, but once a day, and this once in very small quantity.

Milk, tea or sour wines are forbidden, and no food containing pits or seeds are allowed. Fruits, raw or cooked, should be eaten morning and evening; the liberal use of honey and the use of milk sugar instead of cane sugar on your foods is advisable.

At breakfast each morning, instead of a cereal, or with oatmeal, eat a handful of finely cut agar-agar with fresh cream. This can be purchased from the local druggist. During the course of the day eat one to three of the following gems, which should be baked twice a week:

Bran Gems: Two cups of bran, 1 cup of flour, 1 cup of milk, ¾ cup of molasses, ½ teaspoonful of baking soda (dissolved in hot water), ½ teaspoonful of butter, ½ teaspoonful of lard, salt to taste. Bake in a slow oven 45 minutes.

If it is not possible to take the bran gems, take one or two tablespoonfuls of wheat bran cooked in milk with cream and sugar.

At dinner, or before retiring, take a dish of stewed prunes cooked in milk sugar or honey. It may be found that apple sauce sweetened with milk sugar will work as well as the prunes. If so, may alternate this with the prunes, eating one on one day and the other the next.

At certain times each morning and evening make an effort to stool. This should be persisted in even if no success is obtained in the beginning. Its object is to establish a regularity in time. However, at any other time when the desire for stool comes on, respond to it at the earliest possible moment.

One cannot accomplish much worth while in a general treatment of chronic colitis unless individuals have thorough movements of the bowels. This is best accomplished by means of diet rather than by the use of purgatives, however simple, or by any other way such as enemata, trans-intestinal lavage, etc. Those cases having intestinal toxemia require dieting and treatment according to the type of infection, which treatments have already been described.

Where there is disturbance in the endocrine system such as is represented in the long legs as compared to the trunk of the body, kyphosis, cross sex characteristics, eyebrows meeting or being sparse at the outer half, heaviness of the hypothyroid and the excitability of the hyperthyroid, large hands and feet of the acromegalic, retarded sexual development such as is seen in persistency of the thymus or pineal glands, disturbances showing in stained areas in the skin, in that they may suggest deficient suprarenal or thyroid secretion, etc., all require attention along the suggested lines.

The more neurological group are the most difficult to handle because they are commonly recruited from social types of individuals who are more difficult to restrain from doing what they should not or to make to persist in doing what they should. In this group would be required the inforcement of more rest, the cutting out of certain social functions, late hours, discontinuance of certain habits, such as drinking and smoking, a sojourn in the country to relieve them from domestic and financial worries, the use of stimulating baths, perhaps nerve sedatives like bromides or valerian to control the symptoms from time to time, various types of tonics such as iron, strychnia, arsenic, and particularly the constructive types such as are represented in the carbohydrates (malt) and fats, and perhaps the use of nuclein or solutions of strychnia and glycerophosphate hypodermically administered. It is largely this type of individuals who having been so much benefited by a sojourn at a foreign spa, have made this custom such a vogue in America. Of them it may be said that if they lived properly at home they would not require these means, and in such instances where the symptoms make it necessary, the spas in various places in America would serve just as well.

When an appendicitis, gall-stones or any surgical condition (focal infections) co-exists with a condition of chronic colitis it is best that operation be performed before the medical treatment for the colitis is undertaken. One sometimes meets with difficulty in practice in carrying out this policy, but it is distinctly the best in the end. Benefit comes from the operation and then with the added benefit

PLATE XXV

Chronic colitis. Catarrhal condition of right side, irregular contractions in transverse and absence of contractions (peristalses) in left colon. Marked sigmoiditis present. (X-ray by author.)

from careful medical handling of the colonic condition at times the most striking results are accomplished.

As Roberts has brought out, it is sometimes of value in the way of controlling the output of mucus to use a warm 5 per cent. solution of gelatin as an enema, one every day or every other day. Some men have claimed benefit by the use of the so-called intestinal antiseptics. In my experience with this condition, they are useless.

Operative treatment for mucous colitis as represented in appendicostomy, cecostomy, etc., is merely mentioned to be condemned. So far as that method of treatment is concerned, the opening of an appendix and stitching it to the abdominal wall can never drain an intestinal canal, and such as may be accomplished by washing through an opened appendix or a cecostomy opening can just as well be accomplished by the use of the trans-intestinal lavage method of treatment.

SIGMOIDITIS.

In the majority of cases of sigmoiditis there is an inflammatory condition of the mucous membrane in that portion of the gut, also represented in the descending and perhaps the transverse colon above it. Sigmoiditis, however, may exist as an independent condition, the rest of the colon being essentially normal. In these instances the rectal mucosa, particularly the upper part is usually affected. The physiological and anatomical conformation in the sigmoid flexure render it predisposed to all diseases arising from the intestinal tract, and also to those arising from pelvic organs. Being a true reservoir, retaining as it does the fecal matter longer than any other portion of the intestinal canal, it is practically subject to infection from whatever pathological cause is active in the upper part of the tract and also to injury and irritation from any foreign body or irritating substance that passes through the bowel. It is particularly susceptible, therefore, to specific ulcerations such as typhoid, tubercular, dysenteric, etc. These do not represent what is described here as typical sigmoiditis.

The term sigmoiditis was first employed by Mayor, of Geneva, in 1893, and since that time it has been used rather indiscriminately to include catarrhal, ulcerative, interstitial and perisigmoiditic conditions, both primary and secondary.

Catarrhal sigmoiditis appears in both acute and chronic forms. The acute form has very little to distinguish it from ordinary catarrhal enteritis involving more or less of the entire colon. In such instances this portion of the gut is usually most affected. It comes on suddenly

with more or less griping pain, there follow watery passages of mucus with or without blood—in fact there may be some elevation in temperature followed by a period of constipation and irregularity of appetite. At the time the attack is on there is usually considerable distress on pressure or perhaps only subjective pain in the sigmoid region. Most often it is simply a dull aching weight, extending through to the back and the patients sometimes take on a septic look during the acute stage; the tip of the tongue becoming red the complexion muddy, and the pulse somewhat elevated. There is sometimes a thickening or a tumor in the left iliac fossa. Because it is an acute condition there is usually an inflammation of the sigmoid due to irritative putrefactive fecal matter becoming dry. This then causes more irritation and an intense reaction on the part of the mucous membrane with perhaps a furious diarrhea, the movement sometimes passing through a canal in the center of a fecal mass. This may go on until an ulceration ensues, or it may lead to a chronic catarrhal condition with hypertrophy of the cells and of the walls and sometimes contraction of the caliber. Not uncommonly these conditions cause adhesions in the vicinity of the brim of the pelvis on the left side, these adhesions usually leading to obstinate constipation which predisposes to other attacks.

The treatment of the acute attacks consists of cleaning out of the bowel, better with irrigation than with too strong cathartics, limitation of food, ice poultices and rest in bed. An enema containing peroxide of hydrogen serves a good purpose here. The attacks usually subside without much ado but may drift into the chronic catarrhal or even interstitial sigmoiditis, or as stated above, with adhesion formation.

CHRONIC CATARRHAL SIGMOIDITIS.

As has been stated, chronic catarrhal sigmoiditis may result from a recurring acute condition and may be part of a general catarrhal condition, or it may come on in an insidious manner, the sigmoid and upper rectum alone being affected. These patients are always constipated, the stools usually being of hard masses surrounded by mucus. There may be present the type of spurious catarrh mentioned above. Not uncommonly one can feel an impaction in the left iliac region, more or less dullness on percussion of this area, loss of relish for food, palpitation of the heart, muddy complexion, an indefinite type of jaundice, tired feeling, mental weariness, often periods of periodical discharge of mucus with or without the crises of a mucous colic. Such patients often present neurological symptoms.

On proctoscopic examination a pale mucous membrane with sigmoid flexure more or less dilated and sacculated and perhaps acutely flexed may be noted—the latter is best noticed on Roentgenographical examination. The sigmoid is often blocked with a large fecal mass or in the saccules behind the flexures small masses of fecal matter are found coated with mucus and muco-pus. In some instances one may find a small ulcer. Such cases have been designated as mucous or membranous colitis, although this designation is wrong.

CHRONIC INTERSTITIAL SIGMOIDITIS.

Sometimes in cases of chronic sigmoiditis a fecal mass is not the cause of the tumor or the swelling noted on examination. Often in the history of chronic catarrhal sigmoiditis, particularly when there has been acute manifestations, the sigmoid may easily be felt like a small sausage-shaped swelling in the left inguinal region and this may persist for weeks or months. In these cases there is usually a thickening of the walls after each successive attack with a loss of suppleness and a distinct infiltration. It is this condition that has been well designated by Tuttle as an interstitial inflammatory sigmoiditis without pus.

There is always difficulty in defecation, the fecal masses are oval or flat and they contain more or less pus and blood. Generally there is a dull pain in the iliac region which is increased upon the passage of gas or fecal matter. There is more or less inflammation in the mucosa covering of the bowel and to this as well as toxic absorption is attributed the rise in temperature, and the frequent symptoms of peritoneal inflammation. Sometimes this sausage-shaped tumor may rise to considerable distance in the abdomen. As a rule it is fixed and perhaps slightly nodular on its surface. When these nodules are observed one should take into consideration the possibilities of diverticula being present. The proctoscope shows little or no ulceration but suggests more or less contraction of the sigmoid, painful upon pressure and not dilating either to atmospheric or artificial distention.

PHLEGMONOUS SIGMOIDITIS.

Few cases of this condition are on record. They usually occur in the young, and most often in women, the condition being ushered in as an acute attack, inaugurated by a desire to stool occurring frequently, accompanied by rectal spasm, with severe abdominal pain intermittent in character. The condition continues for several days. It is accompanied by slight tenderness over the lower left part of the

abdomen, and occasionally small quantities of bloody serum are passed by the bowels. The end of the muscles on the left side become rigid, and tenderness is marked. Usually a sausage-shaped mass of the sigmoid can be felt. This condition probably is brought about by a neglected chronic type of the condition and on examination at operation a marked infiltration involves the coats of the bowels with the formation of ulcer of the mucosa and pericolonic inflammation; sometimes with the production of pus causing a pericolonic abscess. I have seen one such case of abscess formation lead to the establishment of a general peritonitis.

This phlegmonous process can occur in any section of the gastro-intestinal canal—with the possible exception of the esophagus. The higher up in the digestive tract, the more acute the symptoms, alarming the illness, and fatal the result. The form met with in the sigmoid is the least fatal of the three sites, stomach, small intestine and sigmoid.

TREATMENT.

The chronic catarrhal conditions are best handled by local treatment, directed to a reduction of the inflammation and the removal of the constipation by dietetic means. The lavages best to employ are those containing a weak solution of silver nitrate or fluid extract of hydrastis. In a few cases the use of 5 per cent. gelatin enemata is worth while. Again cases are met with which respond best to a very mild chlorine solution.

With an anti-constipation diet on the one hand and local treatment on the other the majority of these cases clear up. Of the chronic interstitial form the same may be said, excepting here pelvic massage or vibration is often worth an effort. In not a few of these cases the use of a saline cathartic is advisable because saline cathartics in their physiological action, being resorbed by the stomach and upper part of the small intestine and taken up by the blood, are excreted by the mucous membrane of the sigmoid and lower colon. In such instances it is sometimes striking to note the reduction of thickening of the wall of the sigmoid.

One sometimes encounters cases of this sort wherein there is distinct constipation in which it might be advisable to consider operation for the purpose of establishing a passage through the lower end of the intestinal canal. Some of these are those of high fecal impaction—that is, obstruction due to the feces which have become packed above a kink in the sigmoid. Where obstructive adhesions have taken place it often becomes a matter of judgment as to whether

to persist with medical and local treatments or whether to resort to operation. As a rule these cases do not do well treated either way—that is, medically or surgically. In the instance of the establishment of an abscess in the region of the sigmoid, operation is indicated. The same may be said in conditions of phlegmonous sigmoiditis. I have had one case of phlegmonous sigmoiditis with most desperate illness on the part of the patient which required a resection of seven inches of the sigmoid.

It is well in the average chronic case to go slow on operation, because the vast majority of these cases, even when they look quite desperate, clear up. In the presence of acute and grave illness the constant taking of blood counts is advisable for noting the establishment of sepsis in the pelvis. Wherever, on the other hand, there are septic conditions such as the left Fallopian tube or ovary, these require operation.

Lastly, it should be stated that the majority of cases of chronic catarrhal and interstitial sigmoiditis are those of chronic excessive intestinal toxemia and thus it is that careful examination of stools and urines should be made, to see whether this condition exists and if so treatment must be instituted for it. By far the majority of cases of sigmoiditis I have met with are those in which intestinal toxemia was present and which have cleared up when this had been attended to.

References.

1 Boas, I.: Darmkrankheiten, 11, p. 216.

2 Gaultier, René: Les Maladies du Duodenum et leur Traitement, Paris, 1910.

3 Einhorn, Max: "The Diagnosis of Chronic Duodenal Catarrh." Archives of Diagnosis, Oct., 1910.

4 Albrecht: Virchows Arch. f. path. Anat., 156, 285.

5 Codman: Boston Med. and Surg. Jour., 1908, 158, 503.

6 Jordan: Brit. Med. Jour., 1912, 1, 1225.

7 Vanderhoff, Douglas: Jour. Am. Med. Assn., Aug. 18, 1917.

8 Little and Helmholz: Johns Hopkins Hosp. Bull., July, 1905.

9 Cordes: Archiv. f. Pediatrics, 1901.

10 Daniels, Peter: The Proctologist, March, 1912.

CHAPTER X.

Ulcerative Processes.

NECROTIC ULCERATIVE PROCESSES.

The usual form of simple duodenal ulcer will not be considered in this connection. It has special etiology and peculiar clinical features which differ distinctly from the other types of necrotic ulcerative processes. The author believes that the first part of the duodenum in which these ulcers are usually found is essentially a part of the stomach and the consideration of duodenal ulcer can be found in the work on the Stomach where it is dealt with in complete detail.

DUODENAL ULCERATION FOLLOWING EXTENSIVE CUTANEOUS BURNS.

Since Curling in 1842 first called attention to the definite relationship between cutaneous burns and duodenal ulcerations the subject has been of much interest. No very satisfactory explanation of the relationship of the two phenomena has been presented. Why these ulcerations are usually found in the duodenum has never been explained. The general theory is that following the burns toxic substances are secreted with the bile, which, on coming in contact with the duodenal mucous membrane, induce ulceration.

These ulcers differ in many features from the common peptic duodenal ulcer. There may be but one, or there may be from three to six present. They are usually located in the inferior horizontal portion of the duodenum. As a rule they are irregular in outline and perhaps long and narrow, varying in width from 1 to 5 millimeters, and in length from 5 to 15 millimeters. Usually the ulcer develops from the fifth to the twelfth day after the burn—they have occurred as early as the second day, and as late as the seventeenth. They are met with chiefly in young subjects, rather oftener in females than males, and more frequently after burns of the trunk than of the extremities. They are almost invariably fatal, and according to the Fenwicks they occur in 6.2 per cent. of all fatal burns.

EMBOLIC AND THROMBOTIC ULCERS.

The intestines are subject to changes dependent upon alterations of its blood supply just as are other tissues of the body. If a large arterial strand be occluded so as to alter materially the blood supply in a considerable part of the intestines some degree of gangrene is usually the process that takes place, but if smaller branches of the intestinal vessels be blocked, especially those running in the intestinal wall itself, ulceration may ensue. Although thrombosis induced by sclerotic changes in the vessel walls may be the cause of such lesions, a much commoner cause is embolism resulting from valvular disease of the heart, or abscesses or thrombosis elsewhere in the body. Under these circumstances the character of the lesions in the intestinal wall will depend upon the character of the embolism, as to whether it be bland, or infectious. If an embolism includes one of the smaller branches of an intestinal vessel there soon develops a hemorrhagic infiltration of mucous tissues which later extends to the mucosa. The area involved becomes swollen, firm and gray or grayish-red on account of the interference with its nutrition. The tissues of the area of distribution of the vessels soon become necrotic and the mucous membrane with more or less of the underlying tissues is cast off and an ulcer results. Usually the deepest portion of an ulcer is in the center. Frequently the visceral peritoneum corresponding to the area of involvement is the seat of a hemorrhagic infiltration. If the occlusion has been such that the entire thickness of the wall is involved, the ulcer becomes deeper and early perforation may result. Usually, however, the ulcers are of small size and involve only the mucous membrane and submucosa. They may be circular, girdle-shaped or irregular in form. Generally they are multiple. If the embolus be septic the early changes may be the same as in the case of a bland embolus but soon a localized submucous infiltration of leucocytes occurs and a small abscess forms. This increases in size and finally ruptures into the intestinal lumen.

AMYLOID ULCERS.

Amyloid disease of the intestines results from the same causes that produce amyloidosis elsewhere. The condition is most often found in chronic tuberculosis, syphilis, chronic suppuration and the various cachexia. The entire intestinal tract may be profusely affected, but at times the ileum only is the seat of the disease. Sometimes the colon alone is involved. The process may affect any or

all of the tissues composing the intestinal wall, but seldom the mucous membrane itself. The lymphoid follicles usually remain entirely free from the amyloid involvement. The intestines present a pale, shiny, translucent appearance and on application of iodine give a typical amyloid reaction by turning brownish-red, and when subsequently treated with sulphuric acid become blue or violet.

A feature of amyloid disease of the intestines especially mentioned here is the so-called amyloid ulcer. These are ulcers varying in size from that of a pin point to that of the large areas involving the entire circumference of the intestine in a girdle-like fashion, and from 5 to 15 centimeters in length. They have smooth, slightly thickened edges and a base pale with numerous small striations.

According to Colberg they originate from the breaking off of the middle villi by the mechanical effect of the intestinal content. It is probable, however, that the ulceration occurs as a result of the insufficient nutrition of the imperfect epithelium caused by the amyloid degeneration of the underlying vessels.

SYMPTOMS AND TREATMENT.

It is plain that the three above-mentioned types of ulceration are essentially secondary processes. They are characterized particularly by one constant feature, diarrhea, and the character of the diarrhea in any one of them is not distinctive in suggesting the presence of the particular form. The stools may be frequent and watery and usually present the presence of blood. In the case of ulcers following cutaneous burns when blood is met with, a fatal issue usually takes place shortly. The cutaneous burn cases usually are accompanied by considerable pain in the epigastric region, which may not be present with the embolic, thrombotic and amyloid types of ulcer. Strange as it may seem, in the embolic and thrombotic types bleeding is not common, and in the amyloid ulcer it must be expected that when blood is present perhaps some other form of ulceration than amyloid exists. Occasionally in the embolic, thrombotic and the amyloid ulcers no special symptoms, other than the diarrhea, exist.

As was stated before, when duodenal ulceration takes place shortly after extensive cutaneous burns a fatal outcome is the common issue. Really there is very little than can be done to control the diarrhea. Opium may be given, either in the form of morphine subcutaneously or opium powder by mouth. Ice bags may be applied and food withheld for the time being. I have seen one such case recover with marked deformity of the duodenum which required a subsequent gastroenterostomy. After the diarrhea has been on for a short time

the patient loses in strength and vitality very quickly, and then the sustaining measures are in order.

In the embolic and thrombotic types, as well as amyloid ulcers, the treatment of the condition resolves itself into the treatment of the primary disease, and an attempt to control the diarrhea which is best accomplished by mineral and vegetable astringents and opium..

INFLAMMATORY ULCERATIVE PROCESSES.

CATARRHAL ULCERATIONS.

It may be argued that the term "catarrhal ulcers" is inappropriate, but it expresses well enough the nature of a condition of ulceration occurring in conjunction with an apparent catarrhal inflammation seen most often in catarrhal enteritis of rather long duration, and more frequently in children and young adults than in those of middle age or older. The ulcers are present in both the small and large intestine, but more numerous in the latter. They are usually minute, round ulcers with very slight undulating edges; larger ones from confluence of several small ulcers may exist. These areas usually have an irregular outline and one of the features of this type of ulceration is the superficiality of the ulcer. Therefore it involves only the mucous membrane, and generally not even its entire depth. One of the conspicuous features of it is that almost all of the mucous membrane seen in the upper rectum or the sigmoid by an endoscope has been denuded of its superficial cover. At times only small areas of mucous membrane are left between the ulcerated areas, such mucous membrane as is left being in a condition of catarrhal inflammation. The process being very superficial, perforation practically never occurs, but it is not unusual from pathology which extends down in the submucosa, perhaps further, that various degrees of cicatrization may take place.

In my opinion, such types of ulceration are generally due to an infection of the intestinal content, often certain strains of colon bacilli, not dysenteric in type, but which nevertheless have certain dysenteric features. This type of disease was commonly seen by me in the troops which had returned from the southern camps at the time of the Spanish-American War. While it is probable that these strains of B. coli are responsible for the condition it is also general that the bacteriology of the intestinal canal is highly anaërobic and generally has an infection of the B. aërogenes capsulatus. What may be classed as the associated flora of bacillary dysentery is commonly seen present in these cases without the true bacteria of the dysenteric type.

SIMPLE ULCERATIVE COLITIS.

What has been said above is true also in this type of colitis, which represents a more advanced or extensive form. It must be remembered by the reader that these two types of colitis are those in which the dysenteric forms of bacteria are not present. Thus a distinction is made.

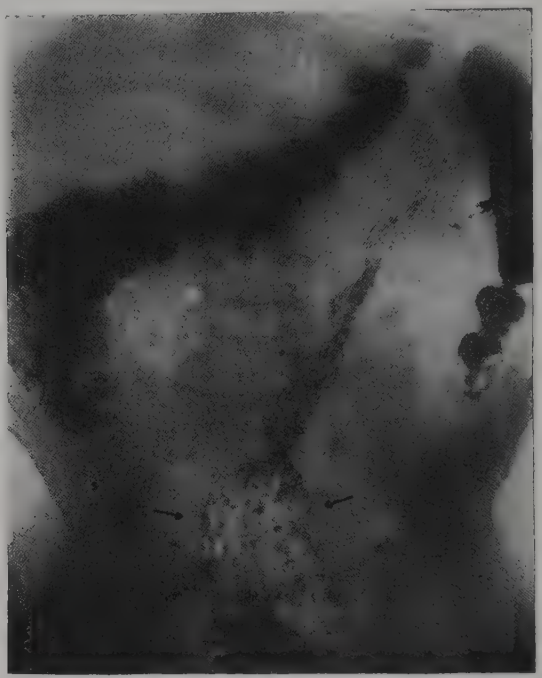


Fig. 66.—Case of ulcerative colitis involving particularly the rectal ampulla. Note flecting on walls of the air distended bowel. (*Case.*)

After rather an extensive experience with these types of ulcerative processes in which considerable laboratory work has been done during the past eight years it is my belief[1] that these types of ulcerative processes are due to a form of B. coli communis. The careful laboratory work in many of these ulcerative processes proves that the B. coli communis can be an infective organism, an infection of the mucous membrane having taken place, the organism gaining entrance into the tissue walls of the gut and living there, probably proliferating. Scrapings from the mucous membrane and the upper rectum and colon in healthy individuals do not show the presence of B. coli

communis in the tissues of the gut wall when proper precautions had been exercised in the scrapings or the examination of tissue. These organisms are Gram-negative, with flagella noted in some of them. They are non-liquefying and they grow readily on all of the culture media, producing organic acids, and readily ferment glucose solutions with the production of acid and gas, and do not produce indol in ordinary nutritive broth. What is striking about them is that the intraperitoneal injections of the bacteria grown from all the cases were fatal within four days to a rabbit, guinea-pig, and cat in each instance, all the animals dying within fifty hours, excepting one cat which lived ninety-three. It is definitely clear then that a pathogenesis present in these organisms is much more fatal than is the case with the usual forms of B. coli communis in normal humans, and inferior animals. It can be proven that the serums from individuals so affected agglutinate their own strain of organisms more definitely than it does the B. coli communis obtained from innocent sources. It is my belief that there is a chronic form of dysentery due to the B. coli communis and that these cases are not uncommon in temperate as well as torrid climates. This organism does not correspond to the Shiga or the mannite-fermenting type, and they are not possible of differentiation from the known forms of B. coli communis of high virulence. These organisms exist in infected cases in large numbers in the lower intestinal tract, mostly in the mucus, but they also can be derived from the substance of the mucous membrane. Either they are definite organisms of the B. coli group or a B. coli communis of a high virulence, strongly hemiparasitic in nature and from which the aggressin production is overwhelming, and against which the body, in susceptible individuals, cannot resist local tissue infection. As a differentiation I personally designate this organism as B. pseudo-dysentericus coli.

As has been stated before, this pseudo-dysenteric type of organism is not the only one that can cause ulceration of the simple type. There are conditions, such as are represented in the saccharo-butyric type of putrefaction, wherein ulceration is due to the high anaërobes, mainly the B. aërogenes capsulatus. Sometimes cases are met with in which the streptococcus fecalis seems to be the infecting organism. It may therefore be said that there are organisms represented in those mentioned, which in susceptible individuals can cause chronic forms of ulcerative processes, which cases fortunately are very amenable to judicious treatment.

FOLLICULAR ULCERS.

Follicular ulceration occurs as a result of the same condition that produces catarrhal ulceration, namely by means of the infecting organisms. Here we see solitary lymphatic follicles mainly involved in the process. Inflammation induces a hyperplasia of lymphatic elements of the follicles which later undergo central softening. Swelling of the lymphatic follicles interferes with the nutrition of the overlying mucosa and subjects it to mechanical irritation so that it undergoes necrosis, and the softened elements of the lymphatic follicles escape, leaving an ulcer. The extent of the follicle is usually greater than the area of superficial ulceration and the resulting ulcer has usually a more or less undermined edge. The ulcers often are numerous, at times giving a typical honeycombed appearance to a part of the bowel. If seen before ulceration has occurred the follicles appear on the mucosa as small shot-like yellowish areas. Perforation seldom occurs. The colon and lower ileum are much oftener the seat of this process than the upper portion of the intestinal tract, and the condition is more common in children than in adults, although adults are by no means exempt.

COLITIS POLYPOSA.
(Virchow.)

It is probable that this is not a distinctive form of colitis with ulceration, but that it may take place as a part of any of the above-mentioned ulcerative processes. In some instances when ulceration has existed for some time, particularly when it is a form of ulceration that extends through the mucosa, islands and tags of mucosa and submucosa take place. Usually these polyps are situated along the line of the attachment of the mesentery, an arrangement that coincides with the blood-supply of the parts. It is probable that the blood-supply of these small and localized areas appeared to have been sufficient to withstand the destructive action of the inflammatory process so that necrosis did not take place, and a productive hyperplasia of the mucous glands and the submucous connective tissue with consequent polyp-like formations ensues instead.

In studying these cases there appears to be first a general colitis, producing a number of local undermining ulcers similar to those that can be found in the above-mentioned forms of ulceration. Such ulcers increase in size, fuse and form large irregular ulcerated areas, and, although the ulcerative process is severe and chronic, it is of such a character that portions of the mucosa and submucosa adjacent

Fig. 67.—Ulceration of follicular colitis, showing the small sized shallow ulcers.

to and supplied by the primary arterial branches are preserved. These portions of the mucosa and submucosa remain as ragged tags scattered over the surface of the colon. I agree with Hewitt and Howard[2] in the consideration of their cases and that the following of their remarks is correct:

"As the process ameliorates and healing begins the irregular margins of these tags become smoothed off and remain as rounded sessile elevations or as polypoid projections of the mucous surface. The portions of the mucosa that remain in these areas regenerate over the surface around the base or pedicle and, if conditions remain favorable, ultimately over the barren and denuded submucosa and together with

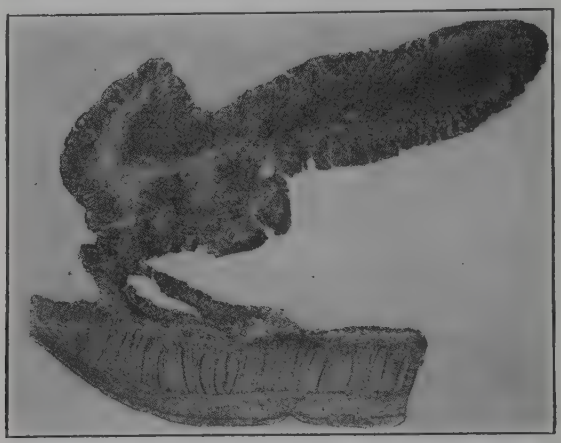


Fig. 68.—From a drawing of a microscopic section of one of the polypi from Woodward's case. The central mass of connective tissue and the surrounding layer of preserved mucosa is well shown.

submucosa over the muscular layer. The mucosa may thus be completely restored, and, with its numerous scattered polypous projections, present a perfect picture of colitis polyposa. Coincident with the process of healing and later, the proliferated fibroblasts begin to contract, as in the cicatrization of a wound. This leads to an occlusion of the orifices of certain of the tubules situated in the polypi and over the mucosa between the polypi. There is then an accumulation of secretion in these occluded tubules with the ultimate formation of retention cysts. These of course increase in size as long as there are secreting cells in their walls, and as there are a greater number of tubules over the surface of the polyp than over the surface of the mucosa, the polyp may appear as a collection of small cysts. It is probably in this manner, as before stated, that the con-

dition which Virchow has designated as colitis polyposa cystica is brought about. It is the end state of colitis polyposa."

STERCORAL OR DECUBITAL ULCERS.

These ulcerations result from the irritative action of fecal masses on the intestinal wall. They occur purely as mechanical results, or what is more probable the mechanical injury to the mucosa and an ingression of pathogenic organisms. The condition is found only in the large intestine, and especially at the point where stagnation of the fecal current occurs—at the hepatic and splenic flexures, or in the rectum, sigmoid, cecum and appendix. These ulcers are usually circular in outline and have an inflamed suppurating base. The condition is more frequently met with in elderly individuals subject to constipation. Perforation of the circular ulcer has been known to occur. Not infrequently these ulcers lead to cicatrization and stenosis. Sometimes these stenoses have been mistaken from carcinoma to stricture, although not uncommonly a carcinoma may develop as a secondary manifestation in the edges of the ulcer, a process similar to development of carcinoma following gastric ulcer.

SYMPTOMS AND TREATMENT.

The symptoms of these types of ulceration may be presented in toto, diarrhea being the main one to draw attention to the condition. This may begin suddenly accompanied by severe abdominal pain, or more rarely, may come on insidiously with a slight looseness of the bowels. Generally within several weeks the stools increase in frequency, and mucus and blood appear. The ordinary remedies as a rule have no effect upon the diarrhea, and the patient rapidly loses weight and becomes ill. The number of stools may vary, commonly from six to eight but as many as twenty to twenty-four in a day are not unusual. The stools usually contain considerable fecal matter, although some of them may be mostly of mucus, pus, blood, water, undigested food and bacteria. One of the features of these cases is that food passes through the alimentary canal with surprising rapidity, sometimes after its ingestion appearing in three hours. Another characteristic of them is that the symptoms are variable, at times present in an acute form, at other times quite subsided. Much depends upon the progress of the disease and the extent of involvement in the gut wall, and particularly its depth. Usually, however, the condition is chronic, extending over years of time. It must be remembered that there are numbers of types of ulcerative colitis in which the symptoms are

never severe and the patients are able to go about although frequently troubled with diarrhea.

Sometimes there is an elevation of one or two degrees of temperature and if a careful record is made of this irregularity it generally suggests a mild degree of septic poisoning. Other cases, however, have no rise in temperature. All observers agree that relapses are very common. When death occurs, which is unusual, it usually takes place from some intercurrent condition or due to exhaustion and wasting, less frequently from perforation and general peritonitis and almost never from hemorrhage.

Usually there is a feeling of general discomfort in the abdomen, particularly when the diarrhea is on; perhaps more or less colicky pains before each stool. In the stercoral type of ulcer the history of constipation is obtained, followed by a diarrheal condition, generally with considerable pain and tenderness on the left side representing the sigmoidal region, or at the flexures. It is probable that some of our cases of massed adhesions of the hepatic colon and also those of adhesions of the pelvic colon on the left side are due to ulcers having been present which have healed with stenoses or pericolonic adhesions, although it is probable that a low degree of inflammation also causes adhesions of the same sort.

What is very important in these cases is to examine the stools bacteriologically to see whether on the one hand ameba histolytica are present, and on the other whether the well-known form of B. dysentericus exists. These forms are not described here. The presence of a diarrheal history such as has been described, and if from an endoscopic examination of the rectum and lower sigmoid ulceration is manifest, effort should be made to gain some idea of the depth and character of the inflammatory process—that is, as to whether it is entirely superficial or whether perhaps it has extended through the mucosa, representing the more resisting type. Careful examinations of the stools are now in order, and this means all that pertains to the bacteriology of the stools that has been advanced in connection with the subject of the saccharo-butyric and the indolic types of intestinal toxemia, and also that which pertains to isolation of one of the forms of B. dysentericus. Where a high anaërobic infection exists a meat diet is in order such as is represented by the following:

The diet is a temporary one. Take mostly meats—all forms of beef with the exception of cuts from the shoulder, kidneys and liver. The same is true of lamb.

The meats should be fresh and taken in a broiled or roasted state. Mutton is permissible, but no pork nor veal.

Take any kind of fish, (broiled, roasted or boiled), with the exception of shad roe and shell fish. Eat eggs in any form.

Butter and whole milk are allowed, together with any form of simple cheese of the cream variety, such as Philadelphia, Neufchatel, etc.

Eat as much gelatin foods as possible.

Oatmeal and rolled oats are allowed.

May have breads or crackers made of gluten or rye flour.

Lentils and dried peas are permissible.

There is no objection to an occasional orange, pineapple or strawberries.

The best drink is chocolate and cocoa.

When on the other hand the condition is due to the pseudo-dysenteric type of colon bacillus or due to the streptococcus, a vegetable diet represents the best form of constant treatment. It is advisable in some of these cases to roughen the diet with considerable cellulose and treat the case as one of constipation. I am not speaking now of stercoral ulcer in which such diet would be definitely indicated, but of the coli infective forms.

It is best to begin the treatment by a rest in bed and the use of large doses of bismuth subgallate, about 4 grams, *t.i.d.* During this time the diet should be bland, consisting essentially of a lactofarinaceous dietary. In the saccharo-butyric type, the protein should be added with caution and only the fluid forms such as eggs, scraped beef, or tender well-boiled fish allowed, and this only after the third or fourth week. Sometimes small doses of an opiate are useful, the best form being Dover's powder.

After many years use of the various forms of rectal irrigation, quinine, weak solutions of lime, etc., I have given up this method of treatment. I believe that when an infection of the colon exists to introduce fluid by rectum tends to cause the infection to mount higher in the colon rather than keep it confined or benefiting it in any local way. In such cases I am not averse to the use of trans-intestinal lavage for the purpose of cleaning out the irritating content of the intestinal canal, in which a hypertonic solution of sodium sulphate and sodium chloride is employed, the irrigations being given twice or three times a week. At the end of a week or two, small instillations of from 4 to 8 ounces of a weak solution of silver nitrate or one to one thousand albargin, or any other form of albuminate of silver may be injected into the rectum, this remaining in over night.

After considerable experience I am in favor, in the coli infection cases, of the use of vaccines. I believe distinctly that it is possible to benefit the condition of affairs in the colon by means of vaccine subcutaneously. In making it, effort should be made to grow as many coli colonies as possible and make a polyvalent autogenous coli.

Because the doses required to beneficially affect the colon usually mean severe local and general reaction, of late I have used lipo-vaccine. This, however, has not given me the benefits that usual vaccine did. These are given every fourth day to a week apart for several months and gradually increased. Lastly it should be mentioned that in these forms of inflammatory ulcerative processes, which may be designated as the non-amebic-non-dysenteric types, patience and length of time are required. Often when one considers the local condition of the patient improved an acute manifestation may take place which is very discouraging to the attendant as well as the patient. It may take years of careful attention before the individual is entirely well. Even then care must be taken that they do not eat injudiciously, do not become chilled in the summer or get too cold in the winter, that they have frequent sojourns in the country and that they live normal lives without excesses, and so on. These individuals usually possess a lowered vitality and a very susceptible colon. Even after the ulceration is healed and has remained healed for a length of time, there is a friability of the mucous membrane which seems to want to break down on the slightest provocation. Therefore it is well to apprise these patients of the fact that they should not be discouraged even at an acute exacerbation, and that it sometimes takes years before a cure is brought about. A few words special may be said to advantage on changes of climate, and the control of daily activities. Not uncommonly those who have lived and had become infected in the south do better if they live for a few years in the north, and those from the east do well in the western part of the country, and *vice versa*. When the avocation of the individual requires him to be active long hours on his feet, this must be modified, or his work changed in character. These two factors are important in the intervals of active therapy, or after the symptoms have been controlled.

In my experience when marked follicular ulcerations have existed which have undermined the mucous membrane, or when polypi are present, a cure is practically never accomplished by medical means alone. It is in these two types of cases that appendicostomy or cecostomy, in addition to the above-mentioned methods of treatment, are usually required. After careful irrigation for a number of months many of these colons take on quite a normal aspect. But again it should be mentioned that after the fistula has healed and no further irrigation from the head of the colon downward is possible, we not uncommonly see a return of the condition. What serves to purpose here is to remember that the medical measures of treatment should be carried out even though an operation has been performed.

INFECTIVE ULCERATIVE PROCESSES.

TUBERCULAR ULCER, STENOSING TUBERCULAR ENTERITIS AND CHRONIC HYPERPLASTIC TUBERCULAR ENTERITIS.

Tubercular ulceration of the bowel may occur as a primary infection, although it usually is secondary. Concerning the relative frequency of primary and secondary intestinal tuberculosis Zahn[3] places the occurrence of primary intestinal tuberculosis at 2.27 per cent., while Ciechanowski,[4] on the basis of 13,203 autopsies, figured it at 1.04 per cent. Zahn found secondary tuberculosis in 63.21 per cent. of all cases of pulmonary tuberculosis. The statistics of other men give it about 50 per cent. Evidently tuberculosis of the digestive tract is more common in children than it is in the adult, and it is not uncommonly found left from other infectious diseases, particularly fevers. However, primary tuberculosis may occur from the ingestion of tuberculous meats or milk. Most of the instances in which intestinal tuberculosis is met with are those of pulmonary tuberculosis in the intestinal lesion usually resulting from the swallowing of the bacillus-bearing sputum. Occasionally, however, secondary tuberculous enteritis can result from the extension of a tuberculous focus in the peritoneum, the abdominal lymph nodes or one of the abdominal viscera.

The most common lesion of intestinal tuberculosis is the ulcer. Other processes may present themselves, either in conjunction with ulceration or independent of it. For the purpose of understanding intestinal tuberculosis best it is profitable to consider separately the ulcerative, the stenotic and the hyperplastic varieties. While clinically the cases cannot be sharply differentiated one from the other, and combinations of the types exist together, at the same time, a proper understanding of the pathological characteristics is best accomplished by a separate consideration of the three varieties because not uncommonly they occur clinically quite definitely as one of the three types.

TUBERCULOUS ULCERS.

The most frequent location of tuberculous ulcers is in the ileum just above the ileocecal valve. They may occur, however, as high as the duodenum and as low as the rectum. The ulcer usually begins in a solitary follicle or Peyer's patch, although it may occur in the mucous membrane itself. It commences as a small gray nodule just below the mucous membrane. This nodule enlarges and undergoes caseating

degeneration in the center. Observed microscopically, it will be seen to consist of a number of typical tubercles composed of giant, epithelioid and lymphoid cells, or of a diffuse caseating mass. Finally the nodule becomes caseous and subsequently breaks through the overlying mucous membrane, the caseous material is discharged into the bowel, and the tuberculous ulcer results. At this stage it has a small, crater-like opening, elevated caseous edges, and a caseous base. Frequently a number of these primary ulcers unite to form a larger ulcer. In other cases the ulceration may begin small, shot-like in size, gradually extending until it becomes an ulcer of considerable dimensions, in which instance there are often considerable miliary tubercles deposited about its margins. Since this extension is by means of the lymph channels, the longest diameter of the ulcer is usually at right angles to the long axis of the intestine, and at times the ulcer extends as a girdle about the entire lumen of the bowel. The margins are elevated, irregular, and usually slightly undermined. Not infrequently these ulcers with tubercles can be seen by the naked eye in the elevated thickened edges. The base appears as a yellowish, caseous mass, or perhaps may be a grayish-red granular area containing numerous tubercles. The peritoneal coat of the ulcer is usually thickened, of a dark bluish-gray color, and frequently contains miliary tubercles, visible as minute, grayish-yellow, shot-like nodules. Also one sees corresponding to the area of ulceration a fine exudation on the peritoneal surface which may lead to adhesions between adjacent coils of the gut. The extent of the ulceration varies greatly in different cases. Ulcers of varying size and age will be found in the same case. Now and then they will become so extensive that small islands of healthy tissue are found surrounded by extensive areas of ulceration.

Complete healing with the disappearance of all tubercles is an unusual occurrence. Not infrequently, however, the ulcers undergo partial organization, so that while some ulceration remains, a moderate degree of stenosis is also present. I have never seen a case of perforation complicating a tuberculous ulcer. The factor which leads to the stenosing of the gut is the thickening of the peritoneal covering and the tendency to the formation of peritoneal adhesions. If perforation occurs, a general peritonitis results, or if adhesions between adjacent coils of intestines have formed, walling off the seat of the perforation, a localized peritoneal abscess occurs. Because of the fact that the ulcers are most frequent about the ileocecal valve and usually most advanced in this area, the site of the perforation is relatively most frequently found in the right iliac fossa, or in the pelvis where

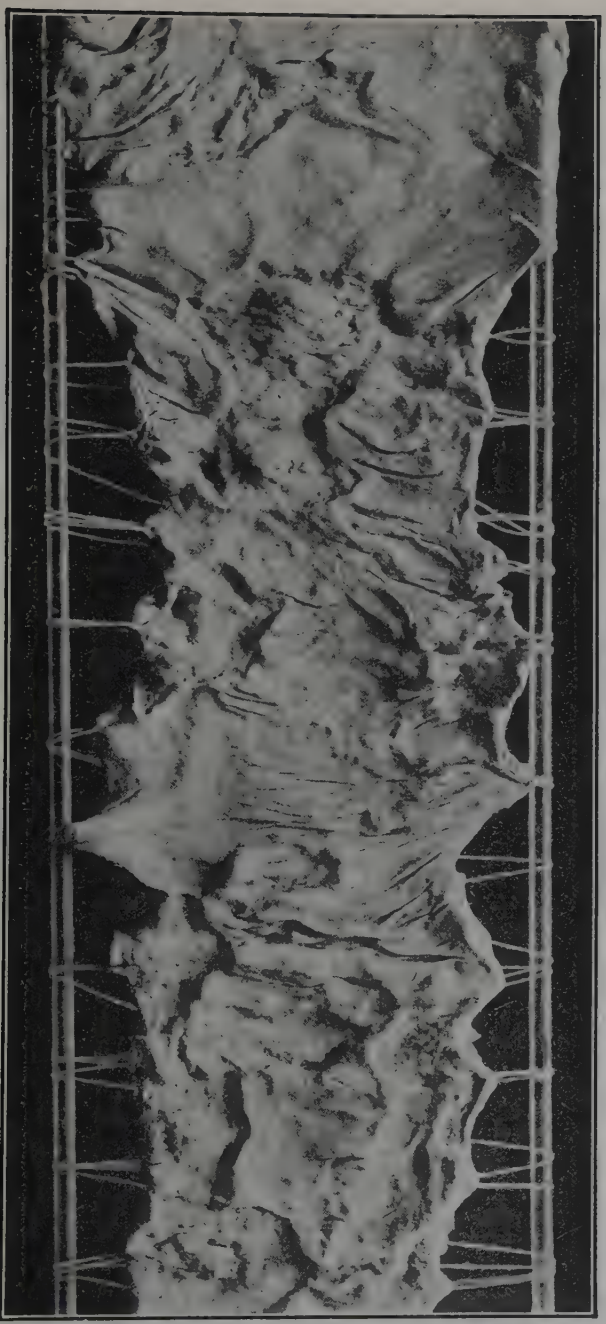


Fig. 69.—Tubercular ulceration of the colon (tubercular colitis). Showing unhealed and healed ulcers.

the coils of the lower ileum usually lie. Ulcers of the rectum not rarely perforate and cause ischiorectal abscesses. It is a well-known fact that many cases of ischiorectal abscesses eventually develop tuberculosis. In this connection it should be remembered that tuberculosis may exist in the body without many if any symptoms, and that a tubercular ulceration of the rectum may take place causing only an ischiorectal abscess, there also being considerable tuberculosis situated elsewhere in the body, particularly in upper levels

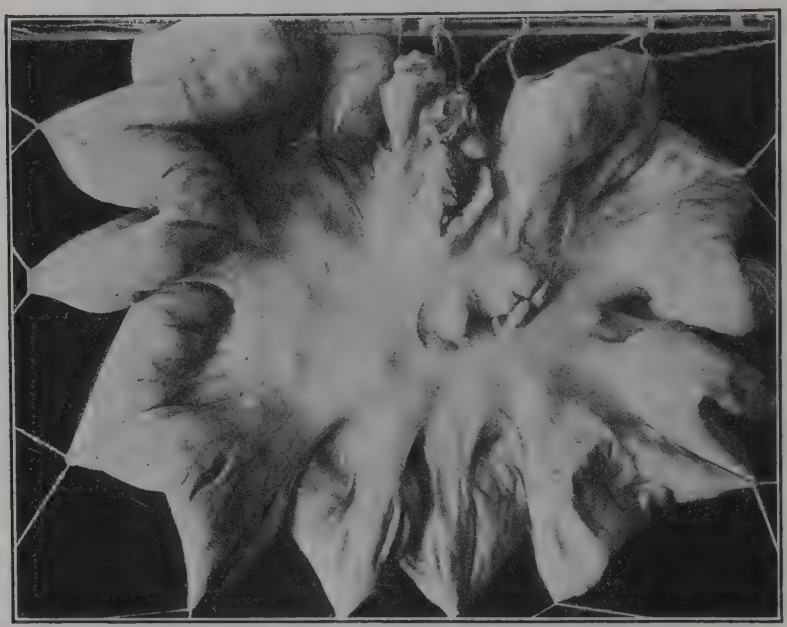


Fig. 70.—Chronic tubercular lymph glands in the mesentery. Largest one at the root of the mesentery.

of the gut. These individuals in the course of time develop symptoms due to the tuberculosis, and it is in that way that an association between ischiorectal abscesses and tuberculosis has taken place. Of course it is manifest that the tuberculosis was present before the ischiorectal abscess occurred. Carcinoma has been known to develop in the site of an old tuberculous ulcer of the intestines.

Symptoms and Diagnosis.—The symptoms of ulcerative, tuberculous enteritis do not differ materially from those of simple enteritis or of other forms of ulceration of the bowels, excepting in the fact that they are chronic. The most constant and characteristic symptom is diarrhea, but this is by no means invariably present even

when there is quite extensive ulceration. When present, the stools are soft and unformed or they may be thin and watery. Mucus may be found in small masses, or as strings or shreds, especially in cases where the feces are semi-solid. Not uncommonly there is an admixture of blood in small quantities and occasionally considerable hemorrhage occurs. When formed stools are passed, or when the feces are hard from constipation, they may be coated with blood-streaked mucus and pus provided the ulceration is situated in the lower bowel. There is probably no one symptom that causes a more rapid emaciation of a tubercular patient than the establishment of a tubercular enteritis with diarrhea. The patient loses strength rapidly, and not uncommonly takes on quite a septic look, this being distinct even through the pronounced pallor which usually exists. A striking feature in these cases is a soreness, localized tenderness which usually is not very marked unless the peritoneum is greatly involved, and which generally is situated in the right iliac fossa. The patients, particularly the young, not invariably complain of a constant soreness in this area. When there is much involvement of the peritoneum considerable distention of the area takes place with a tenderness which reaches as high as the costal margin. Usually the patient's temperature is not much increased by tuberculous enteritis, although it is not uncommon when the tuberculosis of the intestines is well established that the patient runs a low degree of septic temperature.

In every case of marked tuberculosis of the lungs where there is a tendency for looseness of the bowels or the establishment of a diarrhea, the movements of which are generally preceded by attacks of abdominal pain, the abdomen taking on a rounded contour and perhaps some bulging on the right side, always suspect the presence of ileocecal tuberculosis, because as mentioned before, it is present in perhaps 50 per cent. of pulmonary tuberculosis cases whether abdominal symptoms are present or not. To a great extent the associated phenomenon is that a marked pulmonary tuberculosis had been present for some time and that the abdominal condition comes on some time after the pulmonary state. At the same time it must be recalled that amyloid disease of the intestines, the most prominent symptom of which is diarrhea, is a common complication of tuberculosis. The diarrhea of amyloid disease is usually more watery than that of tuberculous enteritis and is less commonly associated with occult or visible blood. In children the diagnosis is usually more difficult to make than in adults. A persistent diarrhea with progressive wasting, abdominal pain and distention and enlarged glands without determinable cause would warrant a tentative diagnosis of

tuberculous enteritis. The diagnosis would be greatly strengthened by the discovery of tubercle bacilli in the stools or a positive tuberculin reaction, but the discovery of tubercle bacilli in the stools is not worth much in diagnosing tubercular enteritis if tubercle bacilli are being swallowed in the sputum.

STENOSING TUBERCULOUS ENTERITIS.

Stenosis of the intestines of tuberculous origin without ulceration results when the tendency to organization as a result of the inflammation exceeds the tendency to destruction of tissue. It is by no means a frequent condition, but it is not uncommonly met with in the gastro-intestinal tract where many cases of tuberculosis are being seen. The tuberculous nature of these strictures is sometimes determined only with the greatest difficulty. I have seen three or four cases in which it was only after a most extensive search of the entire mucous membrane of the intestinal canal that it was possible to determine a tubercular involvement of the gut; of course tuberculosis existing elsewhere suggests a probable cause of the stenosis. The strictures met with are mostly in the ileum, cecum and colon, and what should arouse suspicions of them being tubercular is that they generally are multiple. These strictures in many cases are looked upon as of syphilitic nature, the distinction in both being easily possible clinically, and by the examination of the body at autopsy. I have seen a tubercular stenosis involve only the lower part of the ileum or considerable of the cecum. There is no rule and order. In one case that I had under observation the stenosis involved the last 3 or 4 inches of the ileum and practically a length of about 8 inches of the cecum and ascending colon. In this last case there was no evidence of distinct ulceration, organization having taken place in considerable extension throughout the walls of the gut as well as over a covered area.

The symptoms of these cases are those of chronic intestinal obstruction and the tubercular stage of the condition can only be assumed by the associated lesion or by the exclusion of other etiological factors. When tuberculosis does not seem to exist anywhere in the body, the tuberculin test may be of value.

CHRONIC HYPERPLASTIC INTESTINAL TUBERCULOSIS.

This form of tuberculosis was first recognized by Durant in 1890. The condition is characterized pathologically by varying degrees of stenosis and ulceration, but primarily by a proliferation of tuberculous granulation tissue in the intestinal wall. This leads to a great increase

in thickness of the wall and consequent stenosis, which produces one of the predominating clinical manifestations. The condition may originate in either the mucosa or the serosa, and is usually secondary

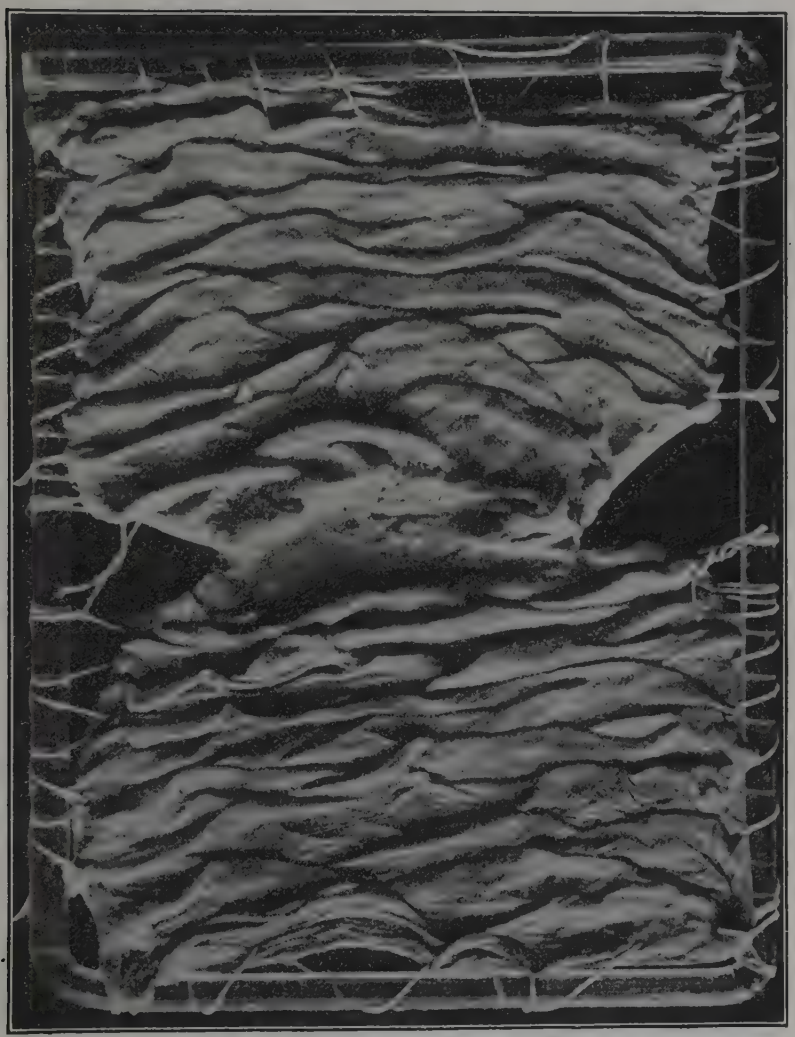


Fig. 71.—Stenosis of the small intestine due to cicatrix in the lumen, gut laid open showing the scar tissue formation and the valvular conniventes. Cause of ulceration probably tubercular.

to tuberculosis elsewhere. It is almost invariably found in the ileocecal region. The predominance of the hyperplastic over the destructive processes is probably a result of decreased virulence of the

tubercle bacillus. The majority of cases met with are between twenty and forty years of age. The active symptoms are preceded by a long duration of vague, rather mild intestinal disturbances. Not until the stenosis is sufficient to produce symptoms of obstruction is the condition possible of diagnosis. I have diagnosed four such cases early by the X-ray. These obstructive symptoms are of insidious onset and form one of the most constant features of the condition. Sooner or later a tumor presents itself, and the resemblance to a carcinoma is often marked. The mass is usually cylindrical in form, giving the impression of a greatly thickened intestinal wall. Occult blood is often found in the stools, and the feces frequently contain visible quantities of admixed blood. Fever is usually an accompaniment of the process.

Treatment.—The prevention of intestinal infection is to some extent possible in the way of controlling the swallowing of infected sputum in tuberculosis cases. It is for this reason that the so-called sputum cup has become so popular, because it is a well-known fact in sanitariums that when patients develop ileocecal tuberculosis the end is usually not far off. Every sanitarium has had the experience of individuals developing ileocecal tuberculosis, thus being reduced to an extreme condition of debility, emaciation, etc. when only a moderate and perhaps very small extent of lesions were present in the chest.

Once the condition has occurred, the main purpose of the treatment is to save the patient's strength by controlling the diarrhea, and as far as possible, the ulceration itself. As has been stated before, there is some tendency of these ulcers toward healing, although usually with our best efforts very little can be accomplished in positive ways. It has been suggested that the diet should be regulated and that this is an essential part of the treatment. After considerable experience in the regulation of diet in these cases I have come to the conclusion that practically nothing can be accomplished by it, and I now allow patients to eat as much as they can and prétty much anything that they desire to take. I have tried all forms of diet and I have never been able to convince myself that any form possesses any value worth the while in these cases. It is a wise thing, however, to remember that raw milk usually does increase the abdominal distress, often with griping pains before movements and not uncommonly the number of movements. Whereas on the other hand, milk which is thoroughly boiled does not do this and seems to act in a beneficial way. Well boiled milk is much better than pasteurized or peptonized milk. I am in favor of having these patients eat three meals a day, taking well boiled milk between times and before retiring.

As far as medication is concerned, very little can be accomplished. One not uncommonly sees in these individuals an almost total lack of desire for food. After employing most every means that I knew of, I have come to the conclusion that from 10 to 20 drops of dilute hydrochloric acid in essence of pepsin given before the main meals answers the best purpose in creating an appetite. It should be remembered that in most all cases of continued fever, and these people usually have more or less fever, if not from the tuberculous condition of the bowels then from the tuberculous condition of the lungs or wherever else it may be situated, a gradual loss of gastric juice takes place. This loss of gastric juice, in my opinion, is due to the effect of the fever upon the secretory apparatus of the stomach. As far as I know I was the first one to point this out. In these instances the use of hydrochloric acid, particularly with pepsin, answers sometimes to very excellent purpose.

Various intestinal antiseptics have been used. Where the diarrhea is excessive, small doses of silver nitrate, acetate of lead, vegetable astringents and others have been employed. It has been said that sulphur seems to exercise a useful effect in some cases. I have used it a number of times and have never seen any benefit from it. So far as medication is concerned I believe that the best results are accomplished by the use of 1 to 2 grain (0.065 to 0.12 gram) doses of powdered opium in about 20 or 30 grains (1.30 or 2 grams) of bismuth subgallate. It is best given in the form of powders and between meals. The patients are permitted to take these according to how the diarrhea is controlled. Sometimes after two or three days the pain in the abdomen has ceased and the movements become rather solid, in which instance the lead powders may be taken, the hydrochloric acid and pepsin before meals being continued.

An important point is that these patients should be kept at rest with as much fresh air as possible. Such indications for the general treatment of tuberculosis are thoroughly in order and one is sometimes surprised at the improvement. It is rare however in cases of extensive tuberculosis of the lung in which there is considerable tubercular ulceration of the bowel that substantial improvement is made for a length of time.

When ulcers are in the rectum direct treatment through the speculum or proctoscope may aid the healing and prevent extension into the perirectal tissues. To this end the use of 5 per cent. silver nitrate solution used every second or third day is worthy of first consideration.

When a stenosis or a marked form of hyperplasia exists with distinct obstruction of the bowel surgical intervention may be called for. Usually all that can be done is an anastomosis, but often striking benefits are accomplished from it.

SYPHILITIC ULCERS.

As must be manifest, initial lesions of syphilis of the intestines are exceedingly rare. These may occur in the acquired case but are most commonly found in instances of congenital syphilis. It seems as though considerable general infection is necessary for the establishment of syphilitic ulcers of the bowel, and it is probable that more cases of syphilitic ulceration of the stomach, and perhaps the duodenum, occur than of the lower end of the gut. The intestinal lesions of congenital syphilis are always associated with syphilitic phenomena in other organs. In the intestines there are found multiple small gummata, almost solely in the small intestines, and especially in the ileum. They take the form of flat, grayish elevations, at times involving the solitary follicles or Peyer's patches, and at times not. Superficial necrosis frequently occurs, leaving an ulcer with a fibrous base and very slightly elevated edges. The ulcers usually extend at right angles to the axis of the bowel, and at times lead to stenosis, and even to perforation.

In acquired syphilis a low degree of enteritis occurs at times. This may go on to the establishment of considerable pathology of the gut due to multiple gummata with the pathology mentioned above. The most frequent situation of syphilitic ulceration of the intestines is in the rectum and particularly in its terminal portion just above the sphincter. The ulcers are distinguished from those of dysentery by their smooth, gray base and the tendency to induration of the edges and extensive stenosis. It is probable that stenosing tuberculous ulcers of the rectum have been frequently mistaken for syphilitic lesions, and *vice versa*. Syphilitic rectal stenoses are more common in women than in men, and according to Bäumler are most frequent between the ages of 17 and 30 years. They may result from the cicatrization of a primary lesion, of mucous patches, or of ulcers that have been caused by necrotic gummata.

Symptoms.—The symptoms of syphilitic involvement of the intestines are those of simple intestinal ulceration or stricture and permit a probable diagnosis only on the basis of the history or the associated findings of syphilis, and possibly their response to the therapeutic test, or to the Wassermann reaction. Syphilitic stenoses of the rectum

Fig. 72.—Syphilitic ulcers of the small intestine. The ulcers are distinguished from the ulcers of the dysenteric type by their shallowness, smooth character of their bases, and the cicatricial induration of their surroundings.

are distinguished by marked induration and extensive cicatrization, and are not difficult of diagnosing, particularly by means of a proctoscope. In the instance of tuberculosis not being found elsewhere in the body and a stenosis existing low in the rectum, particularly in a young woman, one who admits to the performance of the crime of Sodom and Gomorrah, a syphilitic involvement of the rectum should be suspected, particularly when the Wassermann reaction is positive. However, one occasionally meets with extensive syphilitic ulceration of the colon in which a diarrheal history is present. In the three instances of such that I have post mortemed, I have observed that the presence of blood in the stool is not as common a finding as in tuberculous ulceration of the bowel. Usually the history is more chronic and the stools may be semi-solid rather than distinctly fluid. Large amounts of mucus are usually present and by careful searching of the stool one not uncommonly meets with shreds of tissue. This can only be observed when the stools have been diluted and have been passed through a fine sieve, the patient having been kept upon a milk diet for several days before the time of the examination.

Treatment.—The treatment of syphilis of the intestines is directed to the general infection and to the control of the diarrhea. The latter often persists in spite of ordinary treatment and even if active antisyphilitic remedies have been employed. In my experience the best remedy to use in syphilitic involvement of the gut is salvarsan or neosalvarsan in intravenous or subcutaneous injection. The use of mercury salicylate in sterilized oil may be employed. The point to keep in mind is that the treatment must be active and most energetic, or no results will be accomplished. In the presence of stenosis operation may be in order; in which instance it should be remembered that when an anastomosis is to be done most all of the colon may be involved in the syphilitic process, although it does not show on the peritoneal surface, as in cases of tuberculosis of the colon, and that the tissue is subject to be friable and the repair of the stoma may not take place in the normal way or length of time. I am satisfied that I lost one case under operation because extra rows of sutures in the anastomosis were not put in, the gut at *post mortem* having ulcerated through the two rows of sutures, one in the mucous membrane and the protecting row about a quarter of an inch from the edge of the cut. Where a marked stenosis of the rectum is present dilatation might answer sufficiently. I have seen a case which required an incision of the rectum before the trouble with the stenosis could be done away with. Because of the benefit which I accomplished in three cases where there was an extensive syphilitic ulceration of the colon, I am

now prone to a cecostomy being done and irrigation of the colon being established, and kept up for several months at a time. In this instance the use of a weak bichloride solution was very beneficial.

GONORRHEAL ULCERS.

I have seen but five cases of gonorrheal ulcers of the low colon where, in my opinion, infection of the rectum and perhaps the lower sigmoid existed. They were due to infections from without in women who had engaged in the crime of Sodom and Gomorrah and all the cases were identical. There existed quite an active proctitis with considerable thickening of the mucous membrane which was congested, red and bluish in spots, the entire surface being covered by a thin, watery pus. They had a constant leaking from the anus with considerable pruritus and change in the skin about the anus. They did not have diarrhea nor did any of them become stenosed. All were in what may be termed the acute or subacute stage, and it is probable that such a condition as chronic gonorrheal ulceration of the lower bowel does not exist, the bowel being able to throw off the infection with a cure of the case in a length of time. Gonococci were recovered from the pus from smears of the mucous membrane in all instances and the women were hardly aware of their condition exceptiong because of the itching and moisture about the anus.

Because of the danger of carrying the infection upward by the use of any method of irrigating the bowel, even when a recurring method of irrigation is employed, the last of these cases was treated by means of trans-intestinal lavage carried out daily, and topical application of protargol and silver nitrate solution by means of the proctoscope. In the course of a few weeks a complete cure seemed to have been accomplished, and what was interesting was the fact that although she had received admonition one of the cases returned in about five months with a re-infection, suggesting the fact that a gonorrheal infection rather predisposes to the establishment of a second. In this, gonorrheal infections resemble those of diphtheria.

BOTULISM.

Food poisoning of a narcotic type is very rare as compared with the form associated with the term ptomaine. With the latter, intense pain, violent vomiting and exhausting diarrhea, frequently attended by high fever, are the usual symptoms. Contrasted with these are those in which there is little or no pain, no significant temperature, but profound complex disturbances affecting the higher nerve centers. Such

a condition is produced by what is known as sausage poisoning, botulism, or allantiasis, which follows the eating of protein foods decomposed by Bacillus botulinus. A few hundred cases have been recorded with mortality approaching as high as from 10 to 40 per cent. Sausage has been the cause of the great majority, but other articles of diet have been implicated. Among such may be mentioned canned pork, beef, fruit from jars—in fact any form of canned foodstuffs.

It has been stated that the early symptoms come on for the most part at the end of thirty-six or forty-eight hours, the first usually being eye manifestations. Cases are on record, however, in which the symptoms have followed the eating of toxic foods in less than three hours.[5] Sometimes the initial symptoms are those of nausea and vomiting. The initial eye symptoms most often seen are those of the dimming of the vision and diplopia with a tendency of crossage. The ideal case would then present as first of the symptoms occasional mild abdominal cramps with difficulty in swallowing, and perhaps breathing, coming on within a few hours. Usually there is a dryness of the throat, an annoying thirst and a vague feeling of unrest. The voice becomes nasal and the patient begins to feel markedly prostrated. As a rule the bowels are constipated, the output of urine being low. There may be paralysis of the bladder, detrusors or sphincters.

The most striking effect of the condition is that in the nervous system, comprising the symptoms of dimness in vision with a normal background, dilated pupils, ptosis and paralysis of the various eye muscles with double vision and disturbances in deglutition. Dizziness, sleeplessness, marked muscular weakness with no disturbance of the sensorium, no headaches and no anesthesia or paresthesia are characteristic. At times various paralyses of the different skeletal muscles particularly about the neck are seen. Practically all the cranial nerves, except the first and second, have shown involvement in described cases —that is, ptosis, mydriasis, disturbances of accommodation (third and fourth cranial nerves), inability to cry (lacrimal nerve of the fifth cranial), dryness in the mouth (seventh and ninth cranial), disturbance of deglutition partially due to lessened secretion and partially due to nervous disturbances leading to difficulty in chewing and the muscular side of swallowing (ninth, tenth and eleventh cranial nerves), mask-like expression (seventh cranial), at times difficulty in hearing (eighth cranial), variation in speech to aphonia (twelfth), obstipation and respiratory and cardiac disturbances (tenth cranial nerve). The retention of the urine and the occasional paralyses of muscles of the limbs indicate that the spinal cord may be damaged at times. The deep reflexes are not disturbed. Romberg's and Babinski's signs are

both absent. No tremor and no spastic conditions have been described.

As a result of Van Ermengen's classical work on the brain, the question of the pathology of botulismus appears to be well settled. On the basis of the clinical symptoms alone one can assume a specific action of the botulismus toxin on the ganglionic cells of the central nervous system, although other effects of the poison have been observed, namely, a marked paralyzing effect on the gastro-intestinal canal and a tendency to the occurrence of multiple minute hemorrhages, especially in pons and medulla. The former, the paralysis of stomach and bowels, might of course also be ascribed to an affection of the motor ganglia of these organs, either centrally or peripherally. This conception of a specific action of the toxin on the ganglionic cells has been much strengthened by the histological findings in the central nervous system of animals subjected to the experimental intoxication. With these small hemorrhages in the perivascular areas were found. In the case reported by Wilbur and Ophüls[6] there was found a thrombosis of the right arteria vertebralis at lower end of the medulla; the anterior portion of the arteria basilaris was filled with the thrombus. There were thrombi in a few of the pial branches of the basilar artery, in the pial veins at the base of the medulla and pons as well as elsewhere in the brain. All blood-vessels in the brain-tissue were found much distended and full of blood. The thrombi met with were very rich in polymorphonuclear leucocytes, but no bacteria could be demonstrated in spite of the use of the various staining methods. All the thrombi were very rich in fibrin and contained comparatively few masses of conglutinated blood platelets.

Treatment.—As soon as the possibility of botulism presents itself the most evident therapeutic procedure is to empty the stomach thoroughly with a stomach tube, to wash out the large bowel and to leave in the stomach a brisk cathartic—castor oil or Epsom salts. In the two cases that I had under observation, after the initial washing out of the stomach, a trans-intestinal lavage was done which emptied the entire intestinal canal in two hours time, and I feel this had much to do with the diminution of the symptoms, as well as shortening the course of the condition. Since the toxin is the cause of the symptoms and its absorption is slow, the trans-intestinal lavage method with a hypertonic salt solution answers the best purpose. The patient should be kept absolutely quiet, the body supplied with plenty of water, by rectum or under the skin if necessary, simple food given and great care used to prevent aspiration pneumonia. Strychnine seemed beneficial in the cases described in improving the action of the disturbed nervous centers. Other stimulants should be given as required. The

vascular conditions indicate the possibility of complete recovery from a condition that seems hopeless and should lend encouragement to keeping up a hard fight against the disease, even to prolonged artificial respiration in case of respiratory failure.

SPRUE.
(Diarrhea Alba.)

The true etiology of this disease, in my opinion, is settled by Ashford,[7] who first described it in March 1915 under the name of Monilia psilosis. The technique he employed was cultures made from the scrapings of the tongue on glucose agar slants and streak cultures from the feces in glucose-agar plates. Suspicious colonies were plated out for pure cultures on a 4 per cent glucose-agar plus 2 acidity. These plates were incubated for from three to five days. The plate cultures were then examined with a hand glass and all Monilia-suspicious colonies were fished. The organism is a large, bright, round, clean-cut yeast, from 4 to 7 micra in diameter, with at most a few granules and a nucleus. There is usually a pale vacuole in which a violently motile bacillus-like body darts about. The contour is always extremely sharp and well-defined, and it becomes a shell-like envelope in older yeasts. Reproduction takes place by gemmanation. Monilia psilosis always produces mycelial elements.

Diarrhea alba, or sprue, has been known since 1776 when it was described by Hillary of Barbadoes. In literature it is described under different names, such as chronic diarrhea, white flux, cachectic diarrhea, psilosis, and others.

Diarrhea alba, or sprue, is a chronic tropical disease, characterized by the passage of large, frothy, pultaceous, light-colored stools, associated with atrophy of the mucous membrane of the alimentary canal and later of the liver, and raw or ulcerated tongue and mouth, commonly ending fatally after a running and protracted course.

The lesion most met with at *post mortem* is that the tongue is raw-looking and its epithelial coat thinned, while small aphthous ulcers may be found on its tip and edges as well as in other parts of the mouth. The esophagus may show very similar changes, and the stomach may also present minute shallow ulcers and congestive patches. The most important changes, however, are met with in the intestinal canal, where the mucous membrane of the small bowel is thinned, and on section shows extensive atrophy of the villi and tube glands, together with small-celled inflammatory infiltration. These processes are variable in degree, but in chronic cases fibrotic changes are also met with in the mucous coat. The large intestine shows

similar changes in a lesser degree. The liver in the later stages shows simple atrophy and may be considerably reduced in size but only in proportion to the general wasting of the whole body, so that probably this organ presents no distinctive changes.

The above condition appears to be essentially due to a chronic inflammation of the whole lining of the alimentary canal going on to atrophy of the mucous membrane, especially that of the small intestine, and secondarily of the liver, which no doubt accounts for the deficient digestive powers and peculiar stools, and indirectly to the great wasting and eventually anemia of advanced cases of sprue.

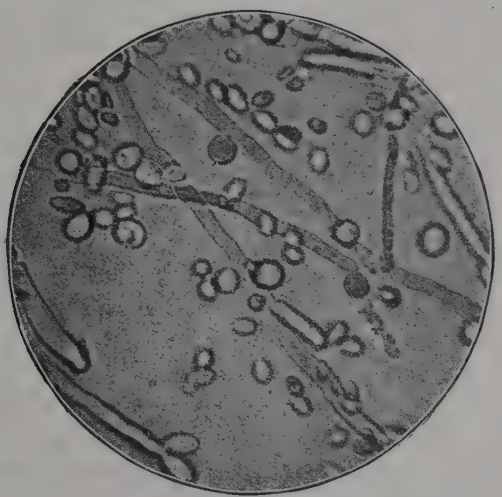


Fig. 73.—Yeasts and hyphaus. Monilia psilosis. (Institute of Tropical Medicine and Hygiene of Porto Rico.)

Symptoms.—This disease is common in the latter part of the year, particularly after the conclusion of a rainy season, although it is met with at all times. By far the largest number of cases are in individuals between 16 and 50 years of age, those between 31 and 50 representing the largest proportion. It may thus be said that sprue is a disease of the prime of life, but in reality it is not absolutely restricted. It is more common among the whites than the mulattoes, and among the true negroes it seems to be rare. Apparently females are about twice as often affected as males.

The onset of sprue is most often that of a severe type of indigestion, regarding which the diagnoses gastroenteritis, acute duodenitis, gaseous indigestion represent the largest number made. Almost a third of the cases, however, have no symptoms at the beginning. In other

cases the symptoms may run on for a while, there being no suspicion of the condition. Gradually, however, a chronic fermentative type of indigestion occurs in which the intestinal symptoms take place usually preceding an infection of the tongue and mouth. The first sign of the disease is generally an irregularity of the bowels, usually thought at first to be a simple type of diarrhea, but tending later to occur in the morning and to become chronic, while the stools become light colored and bulky and the dyspeptic symptoms increase. A steady loss of weight takes place, with perhaps an occasional temporary improvement, sallow complexion, and in the latter stages anemia ensues, and after a longer or shorter course a large proportion of the cases eventually terminate fatally, or in chronic invalidism predisposing to the supervention of terminal acute infections. These usually take years to bring about. It is stated in the study of a number of early cases that the mouth symptoms are absent in about two out of eight cases, while with the advance of the disease this proportion steadily decreases, until in the older cases it is almost always present.

The diagnosis of sprue is not difficult in the typical case, although it is very difficult in the early stages. The success of treatment depends upon early diagnosis, and it is well to look upon any case in a sprue country of chronic diarrhea with light colored stools, very deficient in bile, as likely to be the early stage of the disease. The diagnosis made, such cases should be treated with care. Of course when the mouth symptoms are present they have special diagnostic importance, but their prolonged absence in no way negatives a diagnosis of sprue. The character of the stools usually enables the disease to be detected early, whereby appropriate and timely treatment can be adopted and the disease possibly cut short before it has become a confirmed chronic disorder. It is stated that in the chronic cases at least two-thirds receive no benefit from whatever form of treatment, whereas, those that were carefully treated the first six months of the disease yield much better results. This is important because the rate of mortality in sprue is high, as much as 80 per cent. in the chronic cases.

Treatment.—The treatment of a well established case of this condition is unsatisfactory. A host of remedies have been advocated in the disease, none of which can be relied upon to give good results, and it is a good rule in medicine that when there are many different remedies for a disease none of them are worth much. Of more value is a careful regulation of the diet, and in most cases the fewer medicines given by mouth, the better will be the chances of improvement. An irritating medication generally acts as an excitant and will do more harm

than good to the bare and atrophied lining membrane of the gastro-intestinal tract. Some benefit is found by the use of small doses of ipecacuanha, ⅙ to ⅓ of a grain of emetine hydrochloride, hypodermically given once daily. Arsenic is especially contraindicated, and iron is of no value, even when severe anemia exists.

In the absence of any drug which can be trusted to do any good in sprue, the regulation of the diet becomes the most important line of treatment. A purely milk diet is most generally useful, the quantity being gradually increased until 4 to 6 pints are consumed in the twenty-four hours. It may sometimes be necessary to dilute the milk at first with equal quantities of lime or aërated water and to citrate it. In other cases a meat diet is more successful and should be substituted for milk if the latter is found after a careful trial not to suit the patient. Owing to the deficiency of carbohydrates in meat it is necessary to push the quantity up to 2 pounds in a day, including as little fat as possible as the latter is not easily digestible. Beef and mutton are the best forms, but chicken has often to be relied upon in certain locations. In advanced cases extracts of meat and raw meat juice may be necessary. As improvement in the stools takes place and weight is gained the diet may be cautiously increased by the addition of eggs, fish and some fruit. In certain parts of the world fruit, in addition to milk, has been strongly suggested as a dietary in sprue, grapes having been much given on the continent. Pears, strawberries, bananas, mangoes and other fruits possessing ferments with digestive powers, such as pineapples, have been used freely.

The difficulty in all dietetic treatments of sprue is to induce the patients to continue them long enough to allow of full recovery of the damaged mucous membrane, and until the stools remain formed and contain sufficient bile, weight has been gained, and the symptoms subsided. It is advisable, also, for a patient to be taken to a cold climate, preferably one which is dry.

BACILLARY DYSENTERY.

This condition is of world-wide distribution and is apt to occur in epidemics, thus differing from amebic dysentery, and in the pages of history we have records of many such epidemics. There were marked outbreaks of the condition in the seventeenth and eighteenth centuries in England, and from time to time it is met with in various parts of the world in epidemic form, particularly where sanitary conditions of the country are not good. In Egypt it is very common and it is often found in America, particularly in the southern part of the country. A country today which is most ripe for this condition is Japan, and

it is to the Japanese observers that we are most indebted for much of our knowledge of it.

Agata isolated and described the dysentery bacillus in Japan. This was followed by Shiga's work in 1898, and Kruse's work in Germany. They proved that the bacillus was a specific one and it was later shown that it was the same bacillus. It is known now as the Bacillus dysenteriæ of Shiga and Kruse. Shiga found it in almost pure culture in the blood stained mucus passed in the early stage of acute dysentery. He showed that on recovery it disappeared. Shiga also demonstrated it in post-mortem lesions, and demonstrated an agglutination reaction with the blood serum, and he was enabled to produce dysentery in animals by inoculation with his bacillus. Kruse substantiated these results with his bacillus. Soon after this Flexner also isolated a bacillus in the United States which was the same as the Shiga bacillus. Then there is the Flexner Manilla bacillus which was isolated in new form in the Philippines in 1899, differing both in cultural behavior and agglutination reaction from the Shiga bacillus. This was followed by Strong's discovery of yet another bacillus in the Philippines, and His and Russell's discovery of another which is known as the Y-bacillus, not infrequently is the cause of an asylum dysentery and many epidemics. These are the four ordinarily recognized varieties of bacilli, but in addition there are many others such as Wilmore's El Tor bacillus, which was isolated at the Quarantine Camp; Duval's bacillus, isolated from cases of infantile diarrhea, which differs from the ordinary dysentery organisms by the fact that it ferments lactose; Ohno also described a lactose fermenter.

Organisms have been isolated which have been given the name of para or pseudo-dysentery bacillus, one of which I have described in the foregoing portion of this chapter. It must be remembered, however, that slight differences do not necessarily mean different organisms. They may be only races, and it further has been shown that by prolonged subcultures new sugar fermenting properties can be developed, such differences being usually taken to be characteristic of new species. It is a well-known fact that organisms are temporary and possible of biologic change, particularly when passed through various methods of growth. What is important to remember is that apparently there are four main types of bacilli as having the greatest importance. So far as bacillary dysentery is concerned, the other organisms must be relegated to the background until our knowledge increases.

The bacilli taken as a whole are somewhat short and thick, being about 1 to 3μ. long, they may show variation in shape according to

the culture medium employed and the age of the culture, involution cocci-like forms occurring. None of the dysentery bacilli produce gas. This differentiates them from the coli group. Their action in splitting up the various sugars serves for differentiation among themselves as a group. In a bouillon they all produce turbidity, and after two or three days a precipitate is formed. Indol is formed in cultures of the Flexner Manilla bacillus, but not in cultures of Shiga's bacillus; in the others its appearance is irregular. The growths on agar present appearances similar to gelatin cultures, being white, moist and more or less iridescent. After 24 to 48 hours the round colonies are bluish-grey by transmitted light and whitish by reflected light. All varieties grow on potatoes forming a thin, whitish, rather inconspicuous growth. Litmus milk is not coagulated. All ferment glucose, and none of the common forms ferment lactose. Mannite is the most important sugar, as it divides the bacilli into two groups, the mannite fermenters which form the greater number, and the mannite non-fermenters, represented almost exclusively by the Shiga-Kruse bacillus, which is thus clearly differentiated from the others. The next most impotant sugar is probably maltose, this being fermented by the Flexner bacillus, and not by His's Y-bacillus, and only occasionally by the Strong bacillus. The following is a table showing the principal sugar reactions of the four important bacilli:

	Mannite.	Maltose.	Saccharose.	Dextrose.
Shiga-Kruse	O	O	O	O
Flexner Manilla	F	F	F	F
Strong	F	?	F	O
His's Y.	F	O	O	O

F stands for "ferments" and O for "does not ferment."

Ludke recommends the following method for isolating the bacilli from the feces: "A fresh piece of slime is washed in three lots of sterile water, in order to free it mechanically from adherent feces. Such a piece of slime may be almost a pure culture of dysentery bacilli; it should be as fresh as possible, and is best collected on a piece of sterile gauze, the patient passing it directly onto the gauze. It is then inoculated onto the surface of Conradi-Drigalski's medium in a Petri dish. Lentz recommends that the crystal violet be omitted, as the Shiga-Kruse bacillus grows more readily in its absence (the addition of crystal violet in the proportion of 1 in 10,000 largely inhibits the growth of organisms other than the colon group without materially affecting the Shiga bacillus). The inoculation is best car-

ried out by means of a glass rod with the end bent at a right angle, the mucus being well spread over the surface; two other dishes may be inoculated with the same rod, and then incubated upside down to prevent the condensation of moisture; they should be examined at the end of 24 and 48 hours. Colon bacilli are distinguished by their red color, the acid produced by them acting on the litmus; the dysentery bacilli form blue, dewdrop-like colonies, whereas bacillus subtilis, proteus vulgaris, etc., form very clearly recognized blue colonies with a dry upper surface and a double-contoured border. If a gelatin plate be inoculated the thin colonies of the dysentery bacillus can be easily distinguished from the typhoid colonies. Absence of movement in a hanging-drop culture also distinguishes them from typhoid bacilli. Then, in order to determine the species, they are inoculated into media containing the various sugars. Confirmatory results are obtained by testing the cultures made in bouillon against immune sera."

The Agglutination Test.—After a certain number of days from the commencement of the disease, specific agglutinins appear in the blood, and by means of the Widal reaction a diagnosis of bacillary dysentery can be made, and not only of dysentery but of the particular variety of bacillus as well. This reaction usually does not take place before the seventh day, and it is not often delayed beyond the twelfth day, though in some cases it may not be found before the third week. Information pertaining to the technique of these reactions must be sought in a work on clinical diagnosis.

Shiga in his researches obtained from agar cultures a toxin which produced lesions in the intestine, as a rule without diarrhea, and which produced, in addition, wasting and paralysis. It has been found that a powerful antitoxin could be prepared by the immunization of horses either with the soluble toxin or with the bodies of the bacilli. This toxin is comparatively stable, and is not destroyed by heating at 70° C. for 1 hour, though exposure to a temperature of 80° C. for the same time generally entirely destroys its toxicity. The difference in the susceptibility of different animals to the toxin is very striking. The rabbit and horse are highly sensitive, while the guinea-pig, mouse and monkey are hardly affected.

The antitoxin is capable of protecting animals, either when mixed with the toxin, or when given separately at another part of the body, either at the same time or shortly before or after the toxin. Antitoxin is most readily produced by the Shiga-Kruse bacillus, and, as but little toxin is produced by the Flexner Manilla group of bacilli, this organism probably does not lend itself to the antitoxin treatment, although there are some who believe that it does. A passive immunity can

be produced by the injection of an antitoxin but an active immunity is only produced by a vaccine. It is by such a proceeding that the antitoxin sera are produced. Thus it is that Ludke, bearing in mind Wassermann's experiments with autolysed typhoid bacilli dried and kept in vacuo, which was found to keep well, produced a high degree of immunity with the bacillus of dysentery without much pain or reaction. He prepared a vaccine which he considered superior to the sensitized vaccine.

The Shiga-Kruse bacillus is very well separated from the mannite fermenter and has a definite place by itself, being much more toxic, and not being agglutinated by Flexner's (or other mannite fermenters') serum. It is advised, unless very elaborate laboratory procedures are carried out for the identification of the bacteria in a definite way, that a polyvalent dysentery vaccine be used in the treatment.

Pathology.—In the acute form of bacillary dysentery, the whole of the large intestine may be involved, and the disease may attack the lower part of the small intestine as well, thus differing from amebic dysentery, in which involvement of the small intestine is very rare. Generally the mucous membrane as a whole becomes swollen, red and very vascular. It may be covered by a whitish mucoid exudation, this exudation sometimes resembling the membrane of diphtheria. The edges of the folds may show superficial ulceration or erosion; in a more advanced case the mucous membrane may become gangrenous and large areas take on a greenish-black appearance, and on the separation of the sloughs, serpiginous ulcers are formed. The whole wall of the gut may be thickened, but the deeper coats are rarely involved in the ulceration. There is this radical difference between amebic and bacillary dysentery, that in the former the disease is essentially one of the submucosa and isolated ulcers appear with healthy areas of mucous membrane between them, whereas in the latter the involvement is from the surface, and healthy areas are not seen between the lesions. Extensive sloughing may occur, but though the inflammation and sloughing may spread to the muscular and serous coats, perforation is rare. In convalescent cases healing ulcers are found which leave in many instances a pigmented scar. The disease may in some cases, instead of being completely cured, become chronic; under such circumstances the lower part of the large intestine and rectum are chiefly involved. This is again in marked contrast to amebic dysentery in which it is shown that in chronic or latent intestinal amebiasis the cecum and upper part of the colon are chiefly affected, the rectum being but seldom involved. The ulceration may

last for months, and may end in stricture of the gut. The lesions are best discoverable clinically by sigmoidoscopic examination.

The microscope reveals involvement of the superficial layers of the mucous membrane with fibrinous exudation, small-cell infiltration, and a slight infiltration of the submucous layer. This goes on to loss of substance with destruction and disappearance of the crypts. The muscular coat may also show involvement. In chronic dysentery the crypts may entirely disappear and the floor of the ulcer be formed of granulation tissue. When necrosis takes place, the usual appearance of coagulation necrosis is seen. The mucous on the surface may be crowded with bacilli and may contain many red blood corpuscles.

Symptoms.—The incubation period is generally short, three to six days being the average. The onset is usually sudden, the patient being seized with severe pain and colic in the abdomen. This is quickly followed by a constant desire to defecate. The motions quickly become small and may consist after a while of slight evacuations of blood and mucus. They may number twenty or more in the twenty-four hours. The tenesmus may be very severe owing to the early involvement of the rectum in this form of dysentery. The stools at first do not markedly differ from those of amebic dysentery except that actual hemorrhage is rare, and as a rule there is less blood, but this is by no means constant. The abdomen may be uniformly tender. With such a typical acute case there is usually a rapid rise in temperature. The thermometer may register 104° F. This temperature may persist for several days with fluctuation, and it is seldom as steady as it is in typhoid fever. Toxic symptoms usually rapidly appear, the patient becoming drowsy and listless, especially in the case of children, and exhaustion may set in quickly from the constant pain and tenesmus, the patient being unhappy unless he is constantly on the bed-pan. The pulse rises, the tongue becomes coated with a thick white fur, and may become dry. Albumin may appear in small quantities in the urine. In bad cases vomiting sets in. If the disease persists in this acute form the patient becomes very feeble, with pinched features, dry, inelastic skin, and a small rapid pulse. In favorable cases the symptoms gradually disappear. The temperature becomes normal, and in ten days to a fortnight convalescence is established. Relapses may occur occasionally. In a few cases the condition may become chronic. In the last instance the typical dysentery bacilli may disappear.

A gangrenous form is sometimes seen. In these, after a few days, offensive green or greenish-brown sloughs begin to appear. With these sloughs the toxic symptoms increase, the pulse becomes rapid

and thready, and with a fall in temperature the patient may die. But even in apparently hopeless cases recovery may occur.

A still more severe type of the disease may sometimes be seen in epidemics; this is the choleraic form. The patients are seized with collapse and pass frequent watery stools. They have pinched extremities and present all the appearance of cholera, but bacterial examination reveals the presence of the dysentery bacilli and not the cholera vibrios.

Chronic bacillary dysentery is rare in ordinary civil life but does occur. That most often seen is a direct sequel to the acute form. This is in contrast with the amebic form, which may be chronic from the beginning. There is little or no temperature, but the stools continue to be somewhat frequent. There may be tenesmus with blood and mucus. Emaciation is a frequent sign. It may persist for years, and eventually cure may result, but with a strictured gut and a ruined constitution. The stools sometimes contain in this form bodies like frog-spawn or sago-grains.

In its more chronic form bacillary dysentery is much less characteristic than in its early acute manifestations. Sometimes it is impossible to differentiate it from amebic dysentery by purely clinical appearance. This is due to the fever frequently being absent, or when present, being of a low intermittent type. As a general rule bacillary dysentery terminates in either death or recovery in a few months, and comparatively rarely lingers on with longer or shorter remissions for from one to several years, as is not uncommonly the case with inadequately treated amebic disease. Nor are the remissions so complete and lengthy in the bacillary type, the disease tending to run unchecked until the patient eventually develops some immunity to the infection and slowly recovers. More often, however, the patient becomes worn out by his sufferings and the steady loss of albuminous fluids in the bowel discharges, and succumbs to exhaustion. Extreme emaciation with a retracted abdomen is a striking feature of the clinical picture, but in the terminal stages it may be partly masked by dropsy due to cardiac weakness, atrophy of the general muscular tissue and anemia. Severe hemorrhage from the bowel is less common than in the amebic form.

The two most common complications of bacillary dysentery are hepatitis and arthritis. Hepatitis going on to suppuration is a very common remote complication of amebic colitis, and it might at first sight be expected that acute and chronic bacillary ulceration of the large bowel might not infrequently result in infection of the liver through the portal system. Comparatively it is very rare. Portal

pyemia with multiple small abscesses of the liver may occur as a complication of bacillary dysentery, but this also only rarely happens.

The complications of bacillary dysentery are not numerous. Hemorrhage from the bowel is rare. Heart affections may occur as the result of the toxemia, dilatation of the heart may occur, myocarditis has been recorded, and irregularity with tachycardia. Joint affections may take place, but they are rare. They usually take the form of a troublesome synovitis, usually of the knee, and it is a late toxic manifestation. Irido-cyclitis may exist. Urethritis and rhinitis may occur. Neurasthenia may result, but other nervous manifestations, such as poliomyelitis, are extremely rare. Perforation of the bowel and peritonitis are also rare, and so is liver abscess. If abscess occurs it is of pyemic origin.

The diagnosis consists in the main in distinguishing it from other forms of dysentery. Bacillary dysentery usually has a sudden onset, and there is generally well-marked fever; it usually occurs in epidemic form, whereas with the protozoal dysenteries there is rarely much or any fever, and the onset is not usually so abrupt. In the milder forms, however, of bacillary dysentery this distinction is not marked. The incubation period may be of some help, because in the bacillary forms it is only a few days. The isolation of one of the dysentery organisms makes positive the diagnosis.

The prognosis depends largely on the epidemic. It runs all the way from 10 to as high as 60 per cent. In young children the prognosis is much more serious. However it is possible that with the advent of the serum treatment the mortality is distinctly less.

Treatment.—Unfortunately we have no drug with a specific curative action on bacillary dysentery such as ipecacuanha has on the amebic disease, so apart from the serums and vaccines the treatment is largely empirical.

In the acute cases coming early under observation, the first thing to do is to clear out the bowels with a purge, of which castor oil is the best. After giving about an ounce of castor oil the patient is kept on a fluid diet, composed mainly of citrated milk, during which time the stools are examined bacteriologically. In very mild cases of dysentery treated as soon as the first symptoms appear, it is surprising how quickly all traces of mucus disappear from the watery yellow stools while at the same time relief is afforded to the abdominal pain and tenesmus. To accomplish this then some of the writers who have had the largest experience with bacillary dysentery suggest the use of sodium sulphate purgation after the castor oil for a few days. Opium is often necessary if the stools continue to be frequent and distress-

ing, especially if abdominal pain is present in the intervals between the evacuations. It is best given in the form of an enema at bedtime to ensure some rest during the night, and repeated if necessary in the morning. When administered by mouth it is usefully combined with 1 to 3 grains (0.065 to 0.20 grams) of calomel or given in the form of Dover's powder to counteract its constipating effect. Ipecacuanha is useless in bacillary dysentery except in so far as it exerts laxative properties. The same may be said of emetine. In the acute stage of bacillary dysentery rectal injections are not of as much use as in the chronic forms of the disease. Rogers[8] suggests that the permanganates have an immediate action on the toxins produced by the Shiga bacillus, oxidizing them into harmless substances. For this purpose permanganate solution is made by adding enough permanganate of potash to retain a little of the red color. It is stated by him that the calcium salt of permanganate is preferable to the potassium one, as the former is less irritating to the bowel. Since bacillary dysentery is largely an infection of the colon it seems reasonable to expect a benefit from the use of the trans-intestinal lavage described in which a hypertonic solution of sodium sulphate and sodium chloride is employed. Particularly would this be of benefit in the chronic form of cases. The above measures, together with the gradual establishment of a semi-solid diet answers for the best treatment of the acute form of this disease.

Of late much interest has been attached to the use of serum in the treatment of this disorder, especially in the acute stages. It is made by repeated injections of Shiga bacillus toxins in gradually increasing doses into horses over a long period. The main difficulty with regard to this treatment is that there are a number of varieties of dysentery bacilli in different countries, and different outbreaks in the same country. Thus Ruffer and his colleagues found it necessary to make a polyvalent serum with the aid of dysentery bacilli isolated from cases from different sources, and with it they obtained remarkably good results in the severe and neglected cases among the Mecca pilgrims in which the death rate in bacillary cases was reduced from over 64 per cent. to slightly over 10 per cent. This serum is said to be most effective when administered intravenously.

In established chronic bacillary dysentery the treatment is much more difficult and unsatisfactory than in the earlier stages. The reason for this is because the bacteria have worked their way into the depths of the mucous membrane, the organism culturizing there. One now sees little benefit from the saline treatment mentioned as so useful in the early stages. Frequent trans-intestinal lavage perhaps

with medicated enemata, combined with long careful dieting relieves the irritation produced by the passage of fecal matter over the ulcerated bowel walls and serves the best purpose. It is in this form of bacillary disease that the astringent enemata are most valuable, the best being a solution of silver nitrate of about one pint, the strength of the solution being about ½ to 1½ grains to the ounce. Any of the silver salts may be of use here, even the organic varieties. Copper

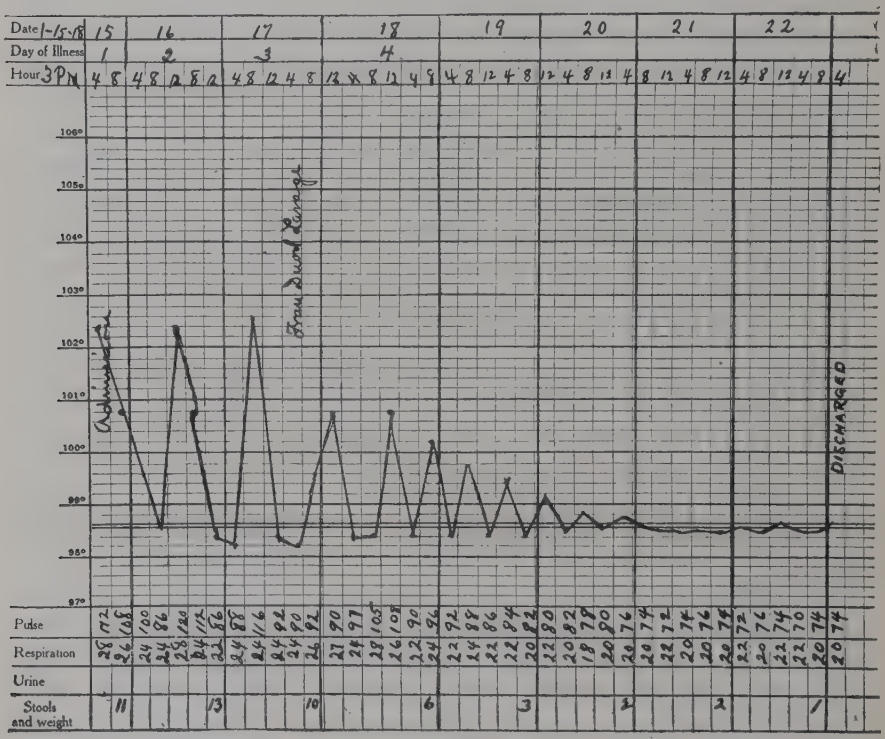


Fig. 74.—Case of ulcerative colitis (bacillary dysentery), treated by trans-intestinal lavage with hypertonic solution.

sulphate in a strength of 1 grain (0.065 gram) to the ounce is often of great value.

Shiga first injected the dead dysentery bacilli as a prophylactic against the disease. Other observers have used similar procedures. The value of vaccine treatment in bacillary disease is still a debatable ground, although the consensus of opinion of those who have had the most experience with it is in its favor. The vaccine most often used is that known as Forster's. The initial dose for an adult should not exceed one lethal dose for a rabbit.

In the chronic forms of the disease dieting is very important. The patient should be kept in bed, or at least free from activity, the diet to be largely a milk diet for a space of time, the milk preferably being boiled. This with large doses of bismuth by mouth, the subgallate salt being preferable, the stools are generally rendered pasty. Slowly the farinaceous foods may be added, preferably those that have been well-boiled, to which the large carbohydrate-bearing vegetables, those having 20 or 30 per cent. carbohydrate content, answers to good service, and such vegetables as potatoes, mashed or boiled, turnips, parsnips, etc., are efficient in sustaining the individual. The stools continuing satisfactory, small quantities of scraped meat or picked fish are to be added. One should be very careful not to add largely of cellulose to the diet and not to resort to protein feeding too quickly. The patient should be kept comfortable and under the best hygienic conditions possible. A prolonged sojourn in the country where the atmosphere is cool and the conditions favorable is often most advisable. There should be freedom from work, excesses of all kinds, mental strain, etc. Great care should be taken that the abdomen does not become chilled, or the general body over-heated. Under ideal conditions with the patient constantly under observation the mortality is low and the patient usually improves, often with small remissions which are easily controlled, and after a few years appears to be well. It is important to mention, however, that many of these individuals seem to have a friability of the colon and under the merest provocation therefor have a remission. Some excellent work in the pathology of the colon of dysentery was done at the time of our Civil War, the disease being prevalent in many of the soldiers. Months and years after the war was over there were pathologies which consisted of more or less superficial cicatrizations and perhaps small ulcerations, generally of the follicular type. With many of these soldiers it took quite a number of years before their intestines were in normal shape, a number of them not recovering completely, whatever the treatment.

DIPHTHERIA.

There have been but few instances reported of a distinct diarrhea assumed to be due to ulcerative processes in cases of marked diphtheria. In a large hospital devoted to the treatment of diphtheria it was very rarely that diphtheritic membrane was found in the gastro-intestinal tract. Occasionally it is formed in the stomach which when seen occurs in limited areas, although it has been known to cover the whole surface. As a rule it is fibrinous, but the hyaline variety is

described. The membrane is always attached to a surface supplied with epithelium and may extend out some distance over apparently normal mucous membrane. Very rarely is a diphtheritic membrane present in the duodenum. Below this point no macroscopic change is ever found in the intestine beyond the swelling of the lymphoid tissue, especially of the lymph nodes. These show the same changes as lymph nodes elsewhere in the body and in the spleen. Whether the diarrhea occurs because of the swelling of the lymphoid tissue or whether it is due to the changes in the liver such as is met with in other acute intestinal diseases, is not known. Undoubtedly the condition is due to toxic substances and not to the presence of the diphtheria bacilli, the well-known lesion of which is a general degeneration, albuminous and fatty degeneration of the liver cells and more rarely necroses which are limited chiefly to the center of the lobule. It therefore is most probable that real ulceration of the enterocolonic tract in cases of diphtheria practically never occurs and such diarrhea as is met with is produced by the effect of toxins upon the lymphoid structures with perhaps the alimentary canal acting as one of the emunctories.

SEPSIS, ERYSIPELAS, VARIOLOID, LEPROSY.

Diarrhea is sometimes met with in instances of the above-mentioned conditions. Generally it is a late symptom and probably is distinctly consequential. Very little is known, and there is very little literature upon the subject as to whether distinct ulceration of the intestinal canal takes place in any of them. It is probable, however, particularly in pyemia, septicemia, and fatal cases of epilepsy, that ulceration may occur. Such ulcerations, of course, would be complications of these diseases and would be very secondary to the diagnosis of the disease itself. The treatment for them would depend upon the severity of the symptoms and how much the symptoms would be influenced in the downward course of the case, remembering that in each one in which ulceration of the entero-colonic canal occurs the case may be deemed as of the lethal type.

PELLAGRA.

The etiological factor in connection with pellagra is still in debatable ground in medicine. Whether it is due to the effect of a chronic intoxication which is caused by silica in colloidal solution in water of determinate composition, and therefore is a disease caused by

colloidal minerals, or whether it is due to some food factor is still not known. The weight of the evidence now seems to bear upon the food factor, although there are many inconsistencies.

All the authorities, ancient and modern, recognize the difficulty of reducing the symptoms of pellagra to a distinct and precise figure, and also recognize the difficulty of making a positive diagnosis of this disease because the manifestations are very different and often are contradictory. In some cases certain symptoms prevail which in others are entirely wanting. There are cases again in which the entire course of the disease is without any characteristic symptomatology, and in which the diagnosis can only be made by exclusion. What is important in the classical case is a triad ascribed to the skin, to the digestive, and the nervous systems. As a rule the onset for the most part is mild and progressive. There comes a certain weakness, even after the least effort, extreme weakness of the lower extremities, an uncertain walk, slow and with short steps, the knees bent and the feet somewhat turned out. The skin is tense, clear and reddened and often has a subicteric tint on the exposed part. There is considerable itching which sometimes becomes aggravated forming vesicles. These break and set free an exudate or may dry as a crust. In other cases there may be pustules or true ulcers. At the termination of the exfoliation, the skin appears somewhat hypertrophic or frequently, atrophic, clear and of a brownish red with whitish winding cicatricial streaks, and specked with telangiectasic spots, becomes inelastic and rolls up in hard wrinkles. The lips are white as if burned with an acid, almost always cracked at the corners and ulcerated. The tongue is red with numerous deep irregular fissures which divide into knolls and give it a muriform appearance, sometimes covered by a thick coating and sometimes completely devoid of epithelium. The eyes are tense, clear with a weak expression and frequently the pupils are unequal. The sufferers have a salty salivation, commonly suffer from an insatiable thirst, loss of appetite, repugnance to food or immoderate hunger. There is a sense of tension and fullness in the stomach region accompanied by burning and irritation. There is severe and frequent pain in the abdomen and a diarrhea which in grave cases becomes profuse and uncontrollable. Vague sensations of heat, formication, numbness of the hands, legs and trunk, buzzing in the ears, pruritis of the body, the hands and feet, headache, darting pains along the neck and spine, and tremors of the limbs, especially the upper, and of the head. The superficial reflexes are either normal or exaggerated; the knee reflexes commonly exaggerated. Frequently there is hyperesthesia, worse on the abdomen or chest. There is an increase of the

moral impressionability and of the psychic excitability, loss of memory, weakness of mind, frequent mutism, sadness, sense of terror, insomnia, religious scruples and a tendency to suicide. The patient often sits doubled up with the chest on the abdomen, the knees against the pelvis and the head inclined. In grave cases the step is tottering, frequently becoming spastic, and paresis or even a true paralysis of the lower limbs occurs. To these symptoms may be added hyperthermia, sometimes albuminuria, and not rarely the ocular lesions, especially blepharitis, keratitis, kerato-iritis, choroiditis and retinitis.

While lesions are met with in almost all parts of the body, only those of the stomach and intestines will be described. The muscular apparatus of the stomach and of the intestine is noticeably reduced in thickness. There is an atrophy or rather an advanced necrosis with extensive vacuolization of the muscular fibers and frequently it is not possible to recognize the normal muscular elements. The glandular elements are likewise destroyed, and not uncommonly infiltrated by small round cells.

The intestines usually are empty and distended by gas or by a greenish-yellow frothy liquid. There may be found a thin and not uniformly but generally a distended tract followed by a restricted area so as to give to the whole intestine a characteristic moniliform appearance. The serosa is almost always hyperemic, especially in the duodenum and the small intestine, and usually presents a hemorrhagic zone of more or less extent. On the other hand, the hemorrhages may be of the punctiform type, not uncommonly seen in one zone and not in another. Hyperemia and hemorrhage are most often found in the cecum and large intestine.

Microscopical examinations of the intestine show the characteristic alterations, especially in the mucosa and musculature. The musculature presents an atrophy and cloudy degeneration; the mucosa a simple hyperemia or a distinct shedding of epithelium, true hemorrhage and a zone of necrosis with intercellular hemorrhagic infiltration.

Symptoms.—One of the best descriptions of the symptomatology is given by Dr. George F. Gaumer of Mexico. He divides the course into three stages. In the first stage there is a sensation of heat in the mouth, throat and stomach; taste is impaired; there is anorexia and frequently ptyalism with a broad, flabby tongue, irregularly marked by red blotches; a peculiar formication in the extremities which often extends very gradually to the whole of the body. About this time small, smooth lustrous specks make their appearance on the dorsal

aspects of the hands and feet. These specks are no longer than a pin's head. They rapidly become more numerous until uniting they form lustrous patches which are checkered off in little squares. These are separated by fine lines giving the cutis a scaly appearance, which is seen better by drawing the skin together with thumb and finger. Sometimes this covers the whole body, but is generally confined to the extremities, chest and back. The skin takes on a senile appearance and the itching becomes almost intolerable, and if scratching is resorted to for relief the burning that follows is unendurable. About this time the patient begins to fail, walks with a heaviness and a peculiarity in his step. He no longer finds his way with closed eyes. The reflexes become greatly exaggerated and his movements are incoördinate. His sleep is much disturbed by hallucinations and strange dreams. During his waking hours he examines himself minutely and with frequency and soon begins to carry on a constant, though inaudible, conversation with himself or some imaginary person. The patient's description of his disease becomes long and tedious, and he often ascribes as a cause of his present condition some insignificant disease or injury that befell him in his youth.

In the second stage the physical sufferings become greater and greater, the skin wrinkles, the appetite fails and this is due to the perversion of the special senses of taste and smell. Hunger increases the mental derangements, and strength fails until the patient is confined to his chair or bed. The mind becomes more deranged as the itching and burning continues to reach his nervous system, so that he often seeks to put an end to his sufferings by committing suicide. The bowels are often constipated, but as the disease advances a diarrhea sets in, which is accompanied by a progressive emaciation, until the patient is reduced to a mere skeleton, or in some cases it passes into a dysentery, which, running a rapid course, soon terminates fatally.

In the third stage we have the final result of a progressive disease going on to a fatal termination. In this stage the mental aspect is deplorable. Fear of impending danger often makes the patient want to flee from home and friends, but unable to do so because of the lack of power of locomotion, his fright increases. These people then begin to suspect everybody as enemies, and finally the patient loses control of the mind altogether and complete dementia generally occurs near the termination of the disease. Such psychoses as are met with are of the melancholic type. Weeks, months and sometimes years are spent in this stage, during which time the patient gradually grows weaker until he is unable to swallow food or drink, to utter an

audible word or to voluntarily move a single muscle, and the spark of life is slowly but surely extinguished.

Treatment.—Until the specific poison which produces pellagra is known no antidote to the poison can be mentioned. It is suggested by Alessandrini and Scala on the basis that colloidal silica is innocuous to the presence of alkaline carbonate and the carbonate of the alkaline earth that effort should be made to antidote the silica by getting into the system carbonate of calcium, magnesium and sodium. For this purpose they have suggested trisodic citrate—(sodium citrate) in a 10 per cent. solution, prepared by dissolving 10 grams of pure neutral sodium citrate in 100 cubic centimeters of distilled water. This is given by hypodermic method in 1 cubic centimeter doses in the deep tissues of the shoulder, back or gluteal region. These injections are given once a day, and after about two weeks given on alternate days. This remedy can be given by mouth in the same 10 per cent. solution, using about 2 cubic centimeters three times a day.

During the course of such treatment, and as is always in order independent of it, tonics and alteratives are to be used. The bowels should be treated so as to control constipation or diarrhea. For this purpose the usual well-known treatments are sufficient.

Excretion by the kidneys must be stimulated, the amount of urine and mineral solids brought up to normal. For this purpose large amounts of fluid are in order. Tonics such as nux vomica or compound syrup of hypophosphates have been suggested. A number of lotions and ointments can be used for the skin lesions. Milk of magnesia, calcium lactate can be given for the acid burning in the mouth and stomach.

PHLEGMONOUS ENTERITIS.

This disease is probably never met with as a primary process. We know that we meet with phlegmonous stomatitis, esophagitis and gastritis, and it is probable that such cases as have been reported as phlegmonous enteritis were really those of phlegmonous gastritis with extension into the duodenum and some distance down into the small intestine. The organism most commonly found in phlegmonous gastritis is a streptococcus, and it is not uncommon in these cases to find that the duodenum has been involved. It is probable that very rarely a purulent enteritis could occur, such being secondary to intestinal ulceration due to other causes or to intussusception, or strangulated hernia. Maragliano has reported septic infection of the ileum, probably by the colon bacillus, with hemorrhage, ulceration and peritonitis.

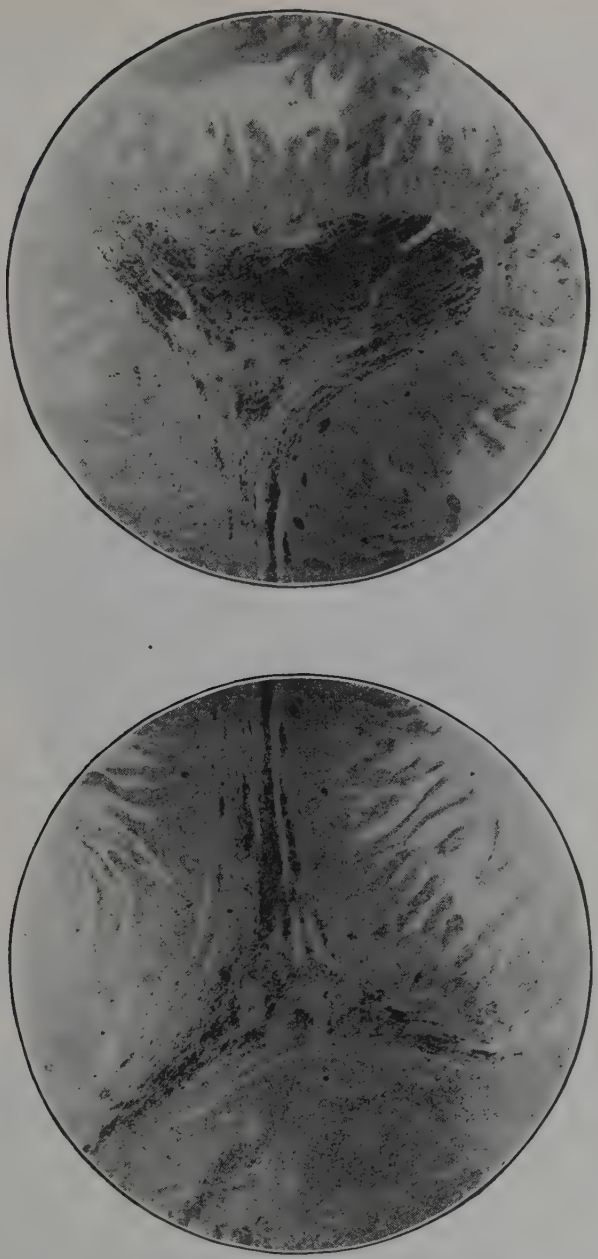


Fig. 75.—Section through valvulæ conniventi of the ileum from a fatal case of phlegmonous enteritis. Note the infective infiltration of the mucosa, the hemorrhage in the tissues of the submucosa and the purulent collection at the base. ×70.

CHOLERA ASIATICA.

This is an infectious disease, caused by the comma bacillus, and characterized by violent diarrhea and rapid collapse.

While present mostly in the East and in India, cases have been met with in America, there having been epidemics in 1832, 1848, 1854, and subsequent to that, quite a few cases having been met with in 1911 from ships from Italy.

The disease is due to a specific organism described for the first

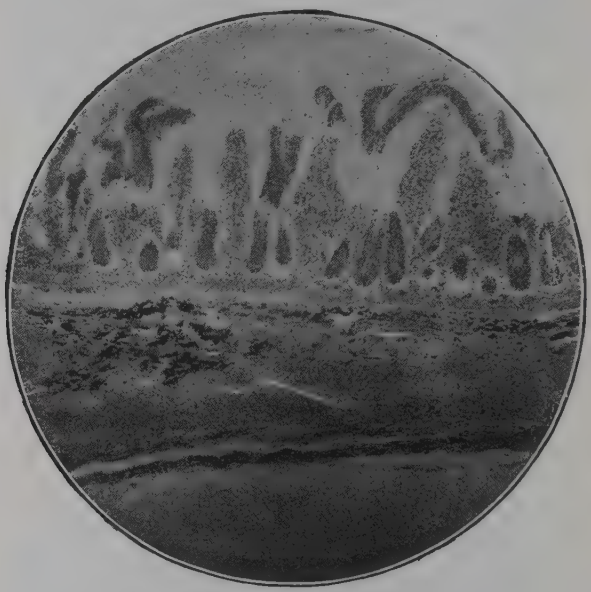


Fig. 76.—Photomicrograph of a portion of the ileum from a fatal case of phlegmonous enteritis. Note the infection, infiltration of the villi, the hemorrhage and thickening of the submucosa and the inflammatory swelling of the peritoneal coat. ×70.

time by Koch, and during an epidemic may even be met with in the feces of healthy persons. In the characteristic case they do not occur in the vomiting, and are met with in post-mortem examinations in enormous numbers in the intestines. They are found in the depth of the glands and in the still deeper tissues. The bacteria has toxic bodies, even in dead culture, and the symptoms, which occur very rapidly, are no doubt also due to an absorption from the intestine when the epithelial layer has been injured.

The disease is not highly contagious, excepting perhaps in the presence of an epidemic when its virulence seems to become enhanced.

It is propagated chiefly by contaminated water used for drinking, cooking and washing, and its dissemination is no doubt due to so-called cholera carriers which are probably even more numerous than typhoid carriers.

Symptoms.—After a period of incubation for from two to five days, the disease sets in with a preliminary diarrhea, in which colicky pains in the abdomen with looseness of the bowels, perhaps vomiting, headache and depression of spirits, without fever, takes place. This diarrhea increases, or it may set in acutely without the preliminary symptoms until profuse liquid evacuations succeed each other rapidly. There may be griping pains and tenesmus. Exhaustion and collapse soon occur, with extreme thirst; the tongue becomes white, and cramps of great severity occur in the legs and feet. Within a few hours vomiting sets in and becomes incessant. The patient goes into collapse, the extremities are cyanosed, and the appearance is that of a dehydration of the body. Usually the surface temperature is below normal while the internal parts may be as high as 103° to 104° F.

At first the feces are yellowish in color, but soon become grayish-white and look like turbid whey or rice-water; whence the term "rice-water stools." In such discharges there are numerous small flakes of mucus and granular matter, and at times blood. The reaction is usually alkaline. As a rule this stage usually lasts for from twelve to twenty-four hours. If a patient survives the collapse, a gradual return to normal condition takes place, although some of them go into a condition known as cholera-typhoid, in which death occurs with coma, the symptoms being attributed to uremia.

Diagnosis.—The only infection with which Asiatic cholera could be confounded is with cholera-nostras—a severe choleraic diarrhea which occurs during the summer months in temperate climates. The absence of an epidemic, the extreme collapse and vomiting with rice-water stools, the cramps, the cyanosed appearance, may be helpful although in severe cases of cholera nostras most of these symptoms may be present. The main distinction in the diagnosis is by bacteriological methods in the discernment of the specific organism in the bowel discharges, there being no specific organism as a cause of cholera nostras. The prognosis is always uncertain, the mortality ranges in different epidemics from 30 to 80 per cent.

Treatment.—Preventive measures are all important. The isolation of the sick and disinfection of discharges have eventually prevented the disease entering various countries and its control in such countries in which it was endemic, such as India and the Philippines.

All fluids should be boiled, errors of diet avoided, and digestive disturbances treated promptly.

In the presence of the condition, however, the patient should be kept at rest in bed, warm, and given a simple diet, boiled milk, whey and egg albumen. Large quantities of water should be given as well as hypodermoclysis to overcome the dehydration due to the diarrhea and vomiting. In the course of the disease, the bowels should be cleansed by castor oil or calomel, and opium is a most efficient remedy to control the diarrhea and pain.

While the author has had no experience with it, the use of the trans-intestinal lavage with a hypertonic solution would seem to be especially indicated in this disease. By this means the intestinal canal could be rapidly cleared much better than by means of castor oil or calomel, and it is plausible to believe that the rate of mortality would be considerably reduced. Irrigation of the bowel with a solution of tannic acid (2 per cent.) in hot water, has been employed with success; the hypertonic solution may be used by rectum. The one advised by Leonard Rogers is composed of sodium chloride, 120 grains; potassium chloride, 6 grains; calcium chloride, 4 grains; water, 1 pint, which he also used intravenously, particularly if the specific gravity of the blood is over 1063, at which time as much as 4 pints may be injected slowly.

INTESTINAL ANTHRAX.

An acute infectious disease caused by the bacillus anthracis. In addition to the cutaneous and pulmonary forms, there is an intestinal. Only the latter will be described here.

It is due to a non-motile, rod-shaped organism, perhaps the best known of all pathogenic microbes. It is a spore-forming bacillus, the main organism of which is readily destroyed, but the spores being very resistant.

In man, the intestinal form occurs by the injection of the organism from the eating of flesh, or drinking the milk of diseased animals, although it may occur from an external infection if the germs are carried to the mouth. The symptoms are those of intense poisoning, the disease ushering in with a chill followed by vomiting, diarrhea, moderate fever, and pains in the legs and back. In the most severe cases there is dyspnea, cyanosis, great anxiety and restlessness, and toward the end, convulsions or spasms of the muscles. Hemorrhages may occur, and often these may be seen in phlegmonous areas or as petechia in the skin as well as in the intestine. The spleen is enlarged. One of the characteristics of this disease is that the blood

is dark and remains fluid for a long time after death, due no doubt to the presence of the anthrax bacillus in the circulation. In the intestinal form the rate of mortality is about 25 per cent.

Two cases of anthrax enteritis have been reported by Brumbaugh, both in young men and both of which were fatal, each one terminating in five days time. The two cases presented the following salient features at autopsy:

Marked involvement of the first portion of the duodenum.

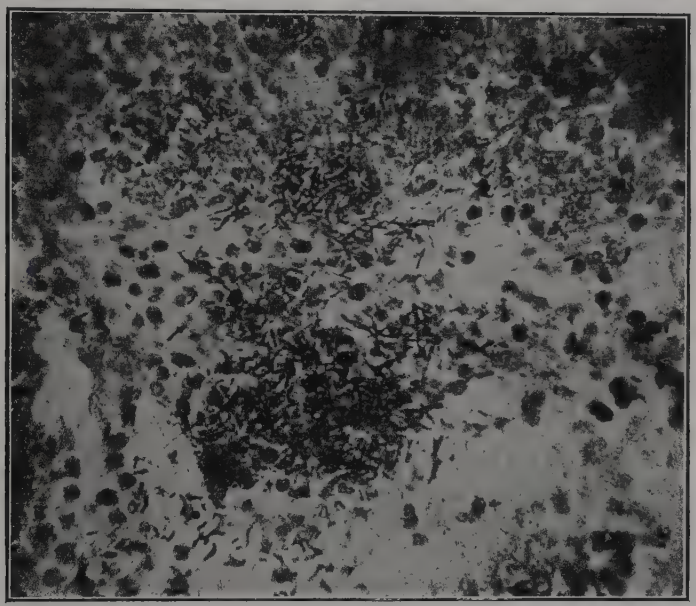


Fig. 77.—Clumps of anthrax bacilli in the submucosa of the intestine. ×480. (*Brumbaugh.*)

Selective action of the infection for the lymphatic tissue of the intestine.

The accumulation of the bacilli in the germ centers of the involved lymph nodes.

Great intensity of the inflammatory process, with associated edema and hemorrhagic extravasation.

Penetration of the entire thickness of the intestine and invasion of the peritoneum, producing seropurulent peritonitis.

Brumbaugh suggested that in a case of infection, sterilization of the patient's hands before meals or the wearing of rubber gloves is a useful measure in the treatment of anthrax to prevent the fatal intestinal involvement.

Treatment.—The most important phase of this is the prophylaxis in which people who are handling hides, hair, rags, etc., should be careful that they do not introduce the bacterium by mouth.

In malignant pustule, the site of the inoculation should be immediately excised, and after the cautery or pure carbolic acid is applied, powdered bichloride of mercury should be sprinkled over the exposed surface. It is important that the local treatment be thorough because these organisms may enter the circulation and be carried into the mouth, and for that purpose it has been advised that subcutaneous injections of 3 per cent. solution of carbolic acid, or 1 to 1000 bichloride of mercury be employed at various points around the pustule and repeated two or three times a day. The internal treatment is confined to the administration of stimulants and plenty of nutritious food. Active purgation in the intestinal case may be given at the onset to remove the infecting material, in which perhaps the use of the trans-intestinal lavage of a hypertonic solution would be most useful. Quinine in large doses has been recommended. Anti-anthrax serum, for which good results are claimed, may be employed.

GASTRO-INTESTINAL INFLUENZA.

This is a pandemic disease occurring at irregular intervals, characterized by the large number of people attacked. It is supposed to be due to a special organism—namely, the Bacillus influenzæ. Numerous pandemics since the sixteenth century have occurred. The disease is highly contagious, and seen in its wildest forms in the cold season of the year.

Symptoms.—The period of incubation is from one to four days, the onset abrupt with fever and its associated phenomena. It is not the purpose here to describe the respiratory, nervous, or so-called febrile forms, but simply the gastro-intestinal.

This occurs with an onset of fever and more or less vomiting and nausea, or the nausea and vomiting may not be present, the attacks being ushered in with acute abdominal pain, profuse diarrhea and collapse. In some epidemics, jaundice, probably due to an extension of a catarrhal process up the common bile duct, has been a common symptom. Usually there is an enlargement of the spleen depending chiefly upon the intensity of the fever, and while this form is supposed to be rare in the United States, I have met with it often enough to say that in an epidemic it is more common than believed. Sometimes in the respiratory, which is the most common, gastro-intestinal symptoms, undoubtedly due to infection of the gastro-intestinal tract,

may be met with, and if occurring when there has been a high fever, severe prostration, or the presence of a pneumonia, it is usually fatal.

Treatment.—Isolation should always be practiced even with gastro-intestinal form, and while no work has been done to recover the characteristic type of bacillus from the stool, it is best to consider the discharge as infective, and thus its sterilization is in order. From the onset the treatment should be supporting, the patient kept in bed, carefully fed and nursed. The bowels should be kept open by the use of calomel, and Dover's powder may be employed to control the distress and diarrhea. The patient should be kept well warmed, and if the fever be high, aspirin given. Where cardiac weakness occurs, stimulants freely given are in order, and during convalescence strychnine in full doses, or the use of thyroid extract. Even after the gastro-intestinal cases the convalescence may be protracted into weeks or months before full health is restored. Thus a good nutritious diet, change of air, and pleasant surroundings are essential. Low spirits and general weakness following this disease is one of its characteristic features.

CONSTITUTIONAL AND TOXIC ULCERATIVE PROCESSES.

SCURVY.

Usually in scurvy the appetite is not necessarily impaired, but septic symptoms may be present as a result of the imperfect diet which produces the disease. Constipation is the rule, but the conditions under which scurvy is developed frequently favor the production of diarrhea of a dysenteric type, and when such a complication exists it may be attended with bloody discharges from the bowel. Very little is known of the pathological condition of the gut present in such instances. It is probable that the hemorrhages characteristic of scurvy cases take place in the mucous membrane, and that with these there is a degree of colitis set up by an infection of the mucous membrane from the intestinal content. Such instances might be accompanied by ulceration, but as is obvious the intestinal condition is dependent upon the constitutional, the treatment of which will not be gone into here.

EMBOLIC AND THROMBOTIC ULCERS.

The intestine is subject to changes dependent upon the alteration of its blood supply. If a large arterial branch be occluded so as to

alter seriously the blood supply of the upper part of the intestine, phenomena will result, discussion of which will be found considered elsewhere in this volume. If, however, smaller branches of the intestinal vessels be occluded, especially those running in the intestinal wall itself, ulceration will often ensue. Although thrombosis induced by sclerotic changes in the vessel walls may be the cause of such lesions, a much commoner cause is embolism resulting from valvular disease of the heart, abscess or thrombosis elsewhere in the body. Under these circumstances the character of the lesion will depend upon the character of the embolism whether it be bland or infectious. If the embolism be small there develops a hemorrhagic infiltration of the submucous tissues which later extends to the mucosa. Swelling takes place and, on account of interference with nutrition, a necrosis, when more or less of the tissue may be cast off, and an ulcer results. The peritoneal surface may be involved with a hemorrhagic infiltration. Usually such ulcers are of small size and very superficial. Generally they are multiple. If they are infected, such as in the septic cases, a localized abscess may occur with or without perforation, although perforation may occur as a part of the dry process. In the infected cases treatment is rather expectant, simply being symptomatic in the intestinal condition. When the condition, however, comes on in an idiopathic way, rest, a minimum diet, the use of small doses of powdered opium and large doses of bismuth, would be indicated. Of course, one should keep in mind the possibility of the development of an ulcer or early perforation, in which latter instance the case becomes operative.

LEUKEMIC ULCERS.

In leukemia, and especially in the more acute form, it is possible for lymphatic hyperplasia in the intestinal wall to occur to such a degree as to induce ulceration of the overlying tissues. Such ulcers are most commonly found in the ileum, although they may be met with in any part of the intestinal tract. The ulcers are of irregular outline and have a ragged uneven base. Microscopically this base consists of a dense mass of lymphocytes. The treatment, of course, is that of the treatment of leukemia, and need not be gone into here.

UREMIC ULCERS.

In a few instances of uremia, ulceration of the intestine may occur. The colon, cecum and lower part of the ileum are the usual seats of such ulceration. The ulcers are generally multiple and for the most

part located away from the mesenteric vessels. They probably originate in the Peyer's patches or in a solitary follicle. Generally they are oval-shaped or may have irregular and map-like outline. The borders are clean-cut and not undermined. Perforation is not an uncommon action. Between the ulcers, the mucous membrane may be injected and it is apt to be pigmented. Healed ulcers have sometimes been found in addition to recent ones.

The treatment of such ulceration is based primarily on the treatment of uremia itself, with added symptomatic treatment of the diarrhea.

TROPHIC ULCERS.

In various nervous and mental disturbances ulceration of the intestines has been observed. Notable among these is that of multiple neuritis, in which neurotic or thrombotic changes in the vessels are found, and it has been assumed by Nothnagel that the intestinal lesions were the result of vascular changes associated with the neuritis. It has also been found that intestinal ulceration occurs in various diseases of the brain and spinal cord, and in a few instances, fracture of the spine, which, if shortly followed by diarrhea, extensive ulceration of the colon should be expected. It is not improbable that these lesions are results of the same factor producing the superficial trophic ulcerations so commonly found in spinal disease.

References.

1 Bassler: Medical Record, October 7, 1911; Interstate Med. Jour., vol. xx, No. 8, 1913.

2 Hewitt, J. H. and Howard, W. T.: "Chronic Ulcerative Colitis with Polyps."

3 Zahn: Münch. med. Wchnschr., 1902, Nr. 2, p. 49.

4 Ciechanowski: Wien. klin. Wchnschr., 1907, Nr. 37.

5 Stiles: Jour. Am. Med. Assn., December 27, 1913.

6 Wilbur and Ophuls: Arch. Int. Med., October, 1914.

7 Ashford, Bailey K.: Am. Jour. Med. Sci., Aug., 1917.

8 Rogers, Leonard: "Dysenteries—Their Differentiation and Treatment."

CHAPTER XI.

Appendicitis.

The fact that the appendix can be removed without causing any demonstrable interference with the alimentary tract or general body justifies the conclusion that the appendix is not a fundamentally necessary organ, although it does not prove that it is functionally inert. Regarding function, attention has been drawn to the large amount of lymphoid tissue in the wall of the appendix and this has caused it to be sometimes designated the "abdominal tonsil." This preponderance of lymphoid tissue, also present in the wall of the cecum, must give to this area some special function with which we are not acquainted. In the herbivora the appendix is a functioning part of the digestive apparatus in that it is necessary for the digestion of vegetable matter. In man it seems to secrete a certain amount of serous fluid and whether this has any definite function or not is not known. It can be definitely stated that whatever function the appendix has it is very minor and its potentialities for evil are greater than its capabilities for good.

Age is somewhat of a predisposing cause in appendicitis. It is most common in individuals between the ages of 10 and 30, although those younger and older are not exempt. Male and female seem to be affected alike. As a disease it seems to be a little more prevalent in the spring and summer than in the autumn and winter. Chronic enteritis, dysentery, typhoid fever, influenza, chronic excessive intestinal toxemia, tonsillitis, rheumatism, various infectious diseases and purpura hemorrhagica are in instances predisposing causes of appendicitis.

PATHOLOGY.

It is only recently that the true nature of inflammation of the right ileac fossa has been determined, and this is due largely to the investigation of diseased appendices removed by early operation. Appendicitis is an infectious process—the consequence of bacterial infection of the appendix.

Anatomically, as well as clinically, two varieties of inflammation of the vermiform appendix may be recognized—an acute and a chronic. Like inflammation elsewhere in the body, the inflammatory manifestations in the appendix may commence acutely or chronically. If it

commences as an acute process, the manifestations may subside for a greater or less interval of time, and the pathological alterations may persist as a chronic inflammation. Thus, chronic inflammation may be the resulting manifestation of a previous acute inflammation, or the condition may begin as a chronic inflammation. The acute and chronic forms of appendicitis can be divided into several varieties, which may be grouped as follows:

Acute Appendicitis:

1. Catarrhal.
2. Interstitial.
3. Ulcerative.
   (*a*) Non-perforative.
   (*b*) Perforative.
4. Gangrenous.

Chronic Appendicitis:

1. Catarrhal.
2. Interstitial.
3. Obliterating.

This classification, which is anatomically correct, indicates the nature of the lesions of the appendix and is not altogether contrary to the clinical course of some of the cases. It must, however, be admitted that the types are numerous during the clinical course and it is not possible to state exactly what the pathology of the appendix is. The reason for this is because a catarrhal inflammation of the appendix may, in a given instance, progress to the interstitial variety. This may in turn be succeeded by ulceration, and even perforation of the organ. Gangrenous appendicitis may also follow in such a train of events.

ACUTE CATARRHAL APPENDICITIS.

By acute catarrhal appendicitis is understood that variety of acute inflammation in which the pathological alterations are wholly or almost wholly confined to the mucous membrane, the other coats of the organ presenting but little or no deviation from the normal. Acute catarrhal appendicitis is probably not uncommon. It gives rise to but few clinical manifestations, particularly if the lumen of the organ be of good caliber. It is likely that this variety of appendicitis constitutes the early stage of many cases of the more severe varieties of acute inflammation, and it is doubtless commonly the starting point of many of the chronic cases.

In this type of appendicitis, the external appearance of the organ is not appreciably altered, although it may feel a little stiffer or firmer

to the touch. The mucous membrane is swollen, hyperemic, and perhaps edematous. The lumen of the organ may be partially or completely occluded at one point or at several points. The contents of these appendices may consist solely of a mucous, turbid, grayish or yellowish-green, or sanguinolent, or there may be a fecal content. When purulent it has been termed purulent catarrhal appendicitis. In such instances slight superficial erosion and desquamation of the epithelium of the mucous membrane may supervene, or, as is not uncommon, there may be hemorrhagic foci in the mucous membrane causing in these diseased appendices what has been designated as hemorrhagic catarrhal appendicitis.

Upon microscopical examination of such appendices the crypts of Lieberkühn are found distended to a variable degree. Usually the contents are of the well-known mucous nature. Some emigrated leucocytes are usually visible between the epithelial cells, and the entire mucosa is the seat of more or less serous infiltration. Beside these alterations and some congestion of the vessels of the mucosa and submucosa, the mild forms of this variety of inflammation of the appendix may present no noteworthy pathological features. If, however, the inflammation be more intense, there is a more severe congestion of the vessels of the mucosa and submucosa, a greater degree of infiltration, a more marked infiltration of the retiform tissue of the mucous membrane with emigrated leucocytes, some cellular proliferation and desquamation of the epithelial cells of the crypts of Lieberkühn and of those lining the lumen of the appendix. Such are the cases that present the most marked instances of the purulent and hemorrhagic types of catarrhal appendicitis.

ACUTE INTERSTITIAL APPENDICITIS.

By this term is designated that variety of acute inflammation in which the pathological alterations extend throughout and involve all the coats of the organ. Sometimes this type of inflammation implicates all the coats of the organ from the outset, but in most instances, it probably begins as a catarrhal process extending from within outward. This type of appendicitis is more common than the catarrhal variety, or at least gives rise to clinical manifestations more frequently.

To the naked eye these appendices appear swollen, edematous, and reddened, and injection of many of the vessels beneath the peritoneal covering can commonly be distinctly detected. The organ is usually quite firm to the touch. The mucous membrane is markedly

hyperemic, edematous and softened, and the entire wall of the organ appears thicker than normal. Occlusion of the lumen in one or more places is common. Appendicular calculi may be found within the lumen of the organ.

What has been stated on microscopical examination of acute catarrhal appendicitis holds good here, excepting that all of the coats of the organ are involved, and that the degree of inflammation is more pronounced. A conspicuous feature of this, as well as of the more severe varieties of appendicitis, is the involvement of the lymphoid element which is commonly swollen, and generally the seat of serous infiltration. The capillaries and lymph spaces appear distended—the former with blood corpuscles, the latter with lymph corpuscles, leucocytes, and sometimes erythrocytes. In some instances small follicular abscesses may be noted. In this type of appendicitis, as well as the ulcerative, one not uncommonly meets with chronic inflammation, strictures, angulations, obstruction of the lumen of the organ, and various pathologies such as in one part a catarrhal inflammation only, in another a marked interstitial inflammation, while in still another part the appendix may seem practically unaffected. Usually the proximal portion is the most likely to present the least deviation from the normal, the tip not infrequently being seriously involved. Some times this tip contains pus and is spoken of as empyema of the appendix. If such an appendix is not removed by operation there occur serous and cellular infiltration and necrosis of the wall of the organ, excessive distention, perhaps perforation and peritonitis, the final process being similar to ulcerative appendicitis.

ACUTE ULCERATIVE APPENDICITIS.

By this term is understood that variety of acute inflammation of the appendix in which there occurs a liquefaction necrosis of the inflammatory exudate and more or less of the wall of the organ in communication with its lumen. This may occur without previous catarrh, such as commonly takes place in septic infection, typhoid fever, dysentery, etc. Two forms may be distinguished—a non-perforative and a perforative, one being merely an aggravation of the other.

To the unaided eye, the appendix is swollen, edematous, excessively congested, and may seem a little firmer to the touch than normal in consequence of the tension of the peritoneal covering. Sometimes the appendix may be as thick as the finger, or thicker, other portions perhaps not being thicker than a lead-pencil or perhaps normal in appearance. Certain regions may present dilatation of the

blood vessels. There may be a diffuse redness, varying in shade from a bright red to a deep reddish-blue or purplish color. If perforation be imminent, the area involved is usually of a brownish-green or blackish-green color, and is softer and more prominent than the adjoining region. It is closely surrounded by an area of intense bright redness, and is usually covered by some discolored exudate. When perforation occurs it usually is round, but it may be ovoid, elongated or without definite outline. In most instances the perforation is very small in size. The contents of the lumen in ulcerative appendicitis vary but slightly. It may be muco-purulent, although most commonly it is distinctly purulent, with more or less admixture of fecal matter, and very malodorous. On the inner aspect marked alterations of the lining membrane and a variable number of ulcers are seen. Sometimes the entire mucous membrane is yellowish-green, much discolored, and resembles a false membrane. When an ulcer is met with it may be single or multiple. The pathological alterations in ulcerative appendicitis are the same as those detailed in catarrhal and interstitial appendicitis but much more exaggerated in degree. There is a marked serous, cellular, and hemorrhagic infiltration of the coats of the appendix, and necrosis may be manifest.

GANGRENOUS APPENDICITIS.

By this term is understood an inflammation or infection of the appendix attended by gangrene. Gangrene of the appendix may arise in one of several ways. It may develop on the basis of one of the previously described varieties of inflammation of the appendix. On the other hand, it may begin with a sudden severe infection of virulent bacteria or their toxins of a previously healthy appendix. This type always leads to peritonitis, and not uncommonly there is considerable extension of gangrenous areas in the cecum, and, as I have seen, in the ileum. It appears as if when these appendices are distinctly involved in gangrene that before they rupture secretion from the peritoneal surface may cause gangrene in other tissues coming in contact with it.

A quarter, a third, a half, or even the entire organ may assume a steady greenish-black color, or it may be swollen, malodorous and softened, and perhaps detached from the remainder of the organ or the cecum as the case may be. The gangrene is that of the moist type. If the gangrene be the result of an acute infection, usually at the end of a short time the entire organ is involved, due to the deprivation of the blood supply. The organ then is increased in bulk, much

softened, and of a characteristic gangrenous odor. In this variety of appendicitis the pathological alterations and the clinical manifestations as well may develop with such rapidity as to merit the designation, fulminating appendicitis, or sloughing of the appendix. Usually pus is present. After spontaneous amputation the appendix occasionally escapes necrosis, being nourished either by the appendiceal artery or by newly formed vessels from the surrounding exudate.

On microscopical examination such appendices present no structure that can be identified with certainty. The less involved portions of the organ present all gradations from moderate interstitial inflammation and ulceration to actual gangrene. Adipose tissue if present may become converted into free fat and fatty acids, the muscle fibers become indistinct and dissolved, and the entire tissue breaks down into a granular and semi-fluid débris.

CHRONIC APPENDICITIS.

In some presumably diseased appendices, particularly in instances of a false diagnosis, the histological alterations are not especially conspicuous. One commonly sees appendices removed on the diagnosis of primary appendicitis in which the pathology present is not more marked than that which can be noted at necropsy from subjects who during life presented no definite indications of appendicitis. In other instances are seen distinct and unmistakable evidences of disease in peritoneal adhesions binding the appendix to various tissues or organs. After considerable experience and observation, I have come to the conclusion that chronic appendicitis is one of the easiest diagnoses of the abdomen to make accurately, and yet it is the one in which more mistakes are made than any other abdominal condition.

CHRONIC CATARRHAL APPENDICITIS.

By this term is understood a chronic inflammation of the appendix in which the pathological alterations are wholly or almost wholly confined to the mucous membrane, the other coats presenting little or no deviation from the normal. These cases usually pursue a mild course, being possibly, now and then, subject to minor acute exacerbations.

On macroscopical examination the appendix seems to be a little thicker, stiffer, and firmer than normal. On incising it, the mucous membrane is found to be of a grayish color and somewhat thickened, the crypts of Lieberkühn being moderately distended, and the mucous membrane covered with a layer of rather thick mucus. The caliber

of the lumen may vary somewhat at different levels, usually as a result of previous attacks of inflammation.

The crypts of Lieberkühn are more or less distended with mucus, and some mucous droplets are also seen in the epithelial cells lining these crypts and the lumen of the organ. It is probable that chronic catarrhal appendicitis does not persist with that pathology, but that it becomes diffuse, constituting chronic interstitial appendicitis.

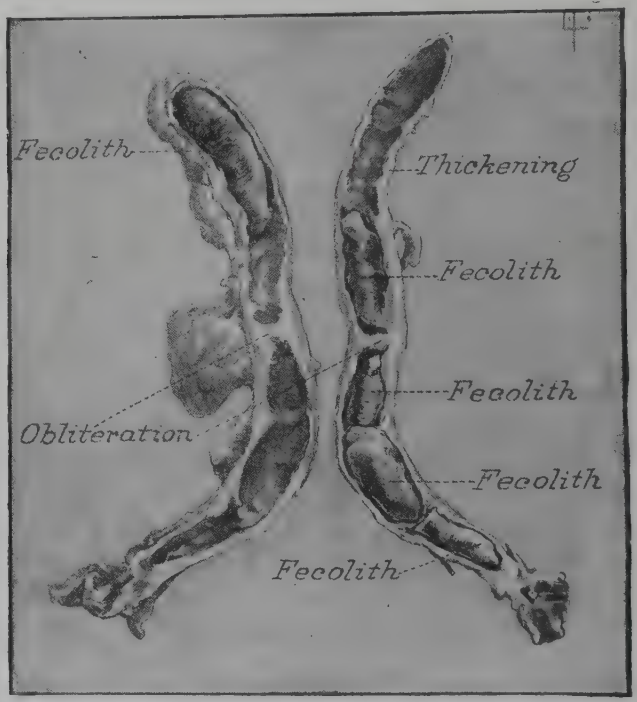


Fig. 78.—Longitudinal section. Chronic thickening of walls of appendix. Impermeable partition in middle. Five fecoliths. (*Pilcher.*)

CHRONIC INTERSTITIAL APPENDICITIS.

By this is understood a chronic inflammation of the appendix in which all the coats of the organ are involved. Certain coats may be involved out of proportion to those of others, but usually there is more or less involvement of each of them. This is the common variety of chronic appendicitis, representing almost all of the properly operated upon cases with that diagnosis.

The naked eye appearances vary considerably in different cases. The simplest form is that in which the condition follows the subsidence

PLATE XXVII

Chronically diseased appendix, showing bulbous formation due to new formed tissue in the walls. (X-ray by author.)

of a minor grade of acute inflammation, or in which the process commences as a chronic inflammation, the lesions not being limited to the mucous membrane. The organ is thicker, stiffer, and firmer than normal and is non-collapsible. The appearance of its mucous membrane and contents do not differ especially from those described in connection with chronic catarrhal appendicitis. At times the lumen is much reduced, varies in caliber in different regions, and contains one or more calculi. It is probable that in instances of moderate degree of acute inflammation, particularly that accompanied by erosion or ulceration, it could under the process of cicatricial healing be replaced by newly-formed connective tissue, which like all newly-

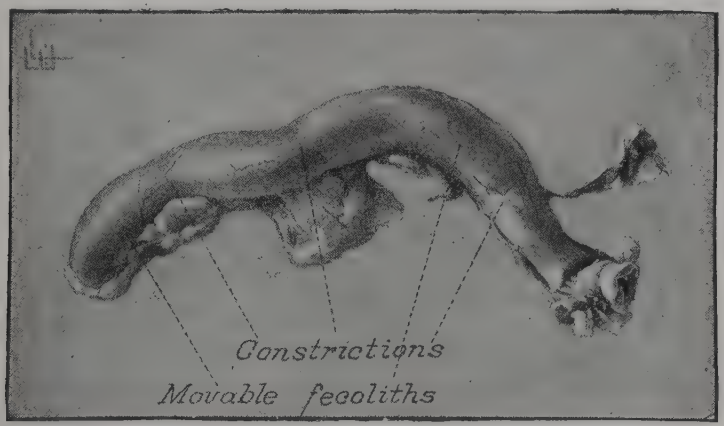


Fig. 79.—Chronically thickened, angulated appendix containing five movable fecoliths. (*Pilcher.*)

formed connective tissue, tends to contract and to form cicatricial tissue. This explains the points of stenosis of the lumen, as well as the angulation, flexure and twist. On the other hand, one may meet with complete obliteration of the lumen of the appendix at one or more points along its course. This is probably due to annular ulceration, granulating surfaces being opposed to granulating surfaces; these adhere, and as the processes of regeneration and organization go on become permanently united by means of newly formed connective tissue. Such conditions not uncommonly give rise to what is known as cystic dilatation, retention, cyst and hydrops of the appendix. Commonly the distal half of the organ is dilated, this being due to obstruction of the lumen.

The microscopical appearance of the appendix in chronic appendicitis varies considerably in different instances. In some the micro-

scopical evidences of disease are very slight. In others the hyperplasia of the submucous and muscular coats is excessive, and there is considerable round-cell infiltration, and perhaps more or less atrophy of the structure. If the appendix be examined while the process of cicatrization of an ulceration is in progress, remnants of the mucous membrane may be detected. The blood-vessel walls are often thickened, and at times there is an interstitial hyperplasia of the nerves, especially of those of the meso-appendix. There may also be some proliferation of the endothelium of the blood-vessels.

OBLITERATING APPENDICITIS.

By obliterating appendicitis is understood a variety of interstitial inflammation of the appendix attended by or leading to obliteration of its lumen, and represents about 2 per cent. of the cases of chronic interstitial appendicitis.

Upon naked eye examination such appendices, as a rule, do not differ materially from those described under the heading chronic interstitial appendicitis. Usually the organ is smaller and stiffer, and it may even present itself simply as a small bud attached to the cecum. Examinations of such appendices commonly present a dense mass of cellular tissue which may be bound down by a mass of fibrous adhesions.

IRREGULAR FORMS OF APPENDICULAR DISEASE.

Among these may first be mentioned tuberculosis of the appendix which may be primary or secondary, miliary or caseous. I have met with three instances of primary tuberculosis of the appendix as far as we were able to judge in a clinical way. It must be remembered, here, however, that tuberculosis commonly exists in a latent form in various parts of the body so that perhaps in each one of these tuberculosis was already present. It is questionable whether primary tuberculosis of the appendix does occur. It is not uncommon to find a tubercular diseased appendix in cases of ileocecal tuberculosis, and as has been mentioned before in the volume, this condition is usually secondary to pulmonary tuberculosis.

Actinomycosis of the appendix is a rare condition, there being but fifty cases reported up to the present time. It can occur as a direct infection through the intestinal tract, or by indirect infection or extension from the thoracic cavity, through the diaphragm, behind the peritoneum, or through the abdominal muscles, or it may be the consequence of metastasis. There seems to be no doubt that it can

occur as a primary affection, the fungus being carried to the appendix by some fragment of carbohydrate substance.

Tumors of the appendix may be primary or secondary, benign or malignant. Those met with are lipoma, myoma, lymph-adenoma, hydatid cysts, fibromyoma and carcinoma, the primary type of which is very rare. In regard to carcinoma of the appendix, it not uncommonly is found in individuals below the cancer age, the age of occurrence having been put by MacCarty and McGrath as thirty years. Practically all such cases are diagnosed and operated upon for appendicitis, the presence of carcinoma being stumbled upon when the abdomen is opened. It seems most probable, as Ribbert has advanced, that malignant growths of the appendix may arise from a partial or complete separation of cells, or groups of cells in instances of chronically diseased appendices. But six cases of sarcoma of the appendix have been described.

PERITONITIS SECONDARY TO APPENDICITIS.

Similar lesions of the appendix in different cases may give rise to most diverse peritoneal lesions. In the majority of instances of appendicitis the peritoneum presents no deviation from the normal. There are many cases, however, even of simple or mild inflammation in which the peritoneum is involved during the acute stage. With the exception of adhesions, in the chronic cases the peritoneum usually seems uninvolved.

In most all types of secondary peritoneal involvement it is surprising that the peritoneum can withstand infection as well as it does. This is due to the bactericidal action of the peritoneal serum, the great resorptive power of the peritoneum, the marked tendency of the peritoneum to unite and form adhesions and cause circumscription—namely, encapsulation of the infectious foci. Peritonitis does result from the access of pyogenic bacteria to the peritoneum when the bacteria are of low virulence or not very numerous. Since in inflammatory conditions of the appendix the number of bacteria are great, the involvement of the peritoneum about the appendix is not uncommon. The disastrous consequences of peritonitis are due to the purulent varieties of the bacteria, to the local suppuration, to the loss of the bodily fluids, and to the intoxication of the general organism, the paramount factor being the last.

The involvement of the peritoneum may be of a serous or serofibrinous type of peritonitis which is most frequently met with. It is found in association with the milder form of appendicitis only—

acute catarrhal, in mild interstitial appendicitis, and the chronic forms of the disease with mild, acute exacerbations. In this stage there is an inflammatory hyperemia of the peritoneum, the vessels being distended and visible to the unaided eye. In addition, the peritoneum has lost its normal luster, is slightly opaque, and is somewhat rough, velvety, or viscid to the touch. There may be small hemorrhagic foci here and there. The fibrinous type is an aggravation or further stage of the serous or sero-fibrinous variety. It is found associated with acute catarrhal and interstitial appendicitis of moderate intensity, and with cases of chronic appendicitis in which there have been recurrences. In this instance there would be a layer of fibrinous deposit of a grayish or grayish-yellow color, and through the medium of this fibrinous or plastic exudate the appendix and the neighboring coils of intestine are more or less firmly united to each other and to the omentum and parietal peritoneum. This serofibrinous type of peritonitis usually does not go on to suppuration but rather to the formation of new connective tissue cells and capillaries, thus causing chronic peritoneal adhesions and bands, the common pictures which we are all acquainted with. It is most probable that by far the largest number of appendices which have adhesions and operated upon in a quiet condition from an inflammatory standpoint, are instances of low degrees of appendicitis with the serous or fibrinous involvement of the peritoneum. The most common peritoneal involvement of acute appendicitis is a circumscribed purulent peritonitis—the formerly so-called perityphlitic abscess. It may follow as a sequence of a serous, a sero-fibrinous, or a fibrinous peritonitis, met with in association with any pathology of the appendix. In circumscribed purulent peritonitis, a greater or smaller portion of the peritoneal cavity lodges a focus of suppuration that develops at the site of the original source of infection and is walled off from the general peritoneal cavity by more or less fibrinoplastic exudate. There may be only a few drops of pus, in which instance the abscess may be difficult to find, while on the other hand, the abscess may be exceedingly large, and may contain upward of a liter of pus. This abscess may be single or multiple, and may be regular or irregular in outline. The wall of the abscess cavity is made up of a grayish-yellow or yellowish-green discolored fibrinopurulent exudate. This is of variable firmness and consistency, and serves to unite more or less securely the appendix, cecum, neighboring coils of the intestine, omentum, mesentery, and parietal peritoneum. The pus is sometimes yellowish in color, but more commonly thinner than cream, of a yellowish-green, brownish or greenish-black color. It usually possesses a peculiar, penetrating, disagreeable, fecal

odor. At times it is distinctly putrid, and may contain gas. With the pus there may be more or less necrotic remnants of fibrinous exudate, one or more fecal concretions, or some fecal matter. The appendix may appear suspended in the abscess cavity, or it may have become entirely separated from its cecal attachment by circular amputation and be floating free in the pus. Most commonly, though, it is found embedded in the exudate forming part of the limiting wall of the abscess.

These abscesses are situated anteriorly, posteriorly, medianly and in the pelvis. The abscess may be retro-peritoneal, even though the appendix be situated intraperitoneally, in which instance it may give rise to extensive phlegmonous infiltration following the course of the iliac vessels and may be present beneath Poupart's ligament. In considerable hospital practice the cases are not few in the course of a year where is found an extensive burrowing of a post-appendicular abscess ascending to the kidney region, perhaps producing an extensive subdiaphragmatic abscess, and every once in a while one meets with such an abscess which has ruptured into the lung.

Beside the matter of infection and the traveling of the abscess, the secondary disastrous consequence is due to the persistency of the abscess causing a lymphangitis, lymphadenitis, thrombo-phlebitis and pylephlebitis. The thrombo-phlebitis usually affects the mesentery and portal veins, but thrombosis of the iliac and femoral veins of either the right or the left side, or of both, may develop. The pylephlebitis is usually mild—the so-called adhesive pylephlebitis —in which the thrombus leads to partial or complete obliteration of the portal vein. Such thrombi may become purulent and lead to the formation of abscess of the liver and subsequently general infection.

In a few instances absorption of the pus may occur. This is rather a rare event, and ensues only when the abscess is very small and the virulence of the contained bacteria very slight. Under such circumstances the abscess gradually becomes sterile, then inspissated, and there may result the formation of a thick mass of indurative connective tissue which rarely has been found the seat of calcareous infiltration.

As a late result there may be found a diffuse or generalized peritonitis involving the entire, or almost the entire, visceral and parietal peritoneum. This form is invariably formed by a small localized process of the right iliac fossa gradually extending and breaking through and involving the general peritoneum and general peritonitis being set up. It is due to gradual leakage of the infectious

material originally at the peri-appendicular site and gradually its continuation and culturization in the peritoneum and leaking between the various coils of the gut. In such instances there is usually not much fibrinous exudate and the entire peritoneum is the seat of an intense inflammatory hyperemia. It is opaque and lusterless, and is covered with a small amount of a grayish-yellow or yellowish-green, veil-like, slimy, viscid exudate. Usually the greatest amount of involvement is on the right side. A type of hemorrhagic peritonitis has been mentioned, in which the peritoneum assumes a hemorrhagic character. There is usually some admixture of blood with the purulent or sero-purulent exudate which gives it a reddish or brownish color.

If an attack of sero-fibrinous or fibrinous peritonitis complicating appendicitis takes a favorable course peritoneal adhesions are formed. The more active the process the firmer and more extensive they are likely to be. In such instances the appendicular peritoneum, usually also the meso-appendix, is congested, thick and opaque and harder than normal. The appendix is more or less firmly united to the cecum, colon, omentum, intestine, mesentery, parietal peritoneum, or some of the viscera, and in women the right uterine adnexa. These adhesions are of varying importance. If they be small, of slight extent, and yielding, they may be devoid of significance. Or, on the other hand, they are commonly distinctly detrimental to health, contributing to renewed attacks of appendicitis by restricting the free motion and the peristaltic action of the appendix, and by causing compression, strictures, twists, angulations, etc. of the organ. In other cases they interfere with the peristaltic power of various portions of the gut, and may even cause intestinal obstruction, angulation, or strangulation.

Bacteriologically the B. coli communis was found by Deaver in about 72½ per cent. in acute appendicitis cases, and 89¾ per cent. in chronic. Other bacteria are met with of which the second most numerous is the staphylococcus pyogenes. Not uncommonly there is a combination of bacteria found. In the ileocecal valve region the greatest number of viable bacteria are met with, thus the danger of infection in a *cul de sac* like the appendix by bacteria gaining entrance into the rich lymphoid structure represents a most plausible cause.

ACUTE APPENDICITIS IN THE ADULT.

Symptoms.—For the purpose of presentation, the symptomatology of appendicitis will be offered under the heading acute which includes exacerbations of chronic appendicitis. There are four cardinal symp-

toms of acute appendicitis, pain, rigidity, nausea or vomiting, and leucocytosis. So far as the average case of acute appendicitis is concerned it may be said that the mistakes met with in diagnosis are those in which one or more of these cardinal signs are missing. If the symptoms are all present the diagnosis is unquestionable.

Pain is the initial symptom in all cases. It usually develops suddenly in one who has previously been well, continues a length of time, recurs at irregular intervals, and is usually cramp-like or colicky in character. Whether the cramp-like character is due to appendicular colic or not is unknown, but as Deaver has said, there is no doubt that its paroxysmal character is one of its characteristic features. The degree of pain bears a direct relation to the severity of the inflammatory process and the variations in the sensibility of different people to pain. After a while the paroxysms gradually lessen in number and severity, although in some cases it is continuous of a moderate severity, increased by coughing or deep breathing, and its subsidence means either the relenting of the attack or the presence of perforation or gangrene. In some few cases the pain is non-paroxysmal, constant and dull from the very onset. This is particularly true in recurring attacks, and the pain is described of a peculiar boring character. The pain is usually situated in the umbilical region, or in the epigastrium, and least commonly in the right iliac fossa. If the appendix be long and overhangs the brim of the true pelvis the pain may be referred to the left side of the abdomen, to the region of the ovary in the female, or along the course of the spermatic cord toward the testicle in the male. If the appendix is post-cecal with a diseased tip and points upward the pain may be referred to the loin or back or to the region of the kidney or liver.

Tenderness on pressure is one of the most valuable signs of acute appendicitis. The area of the tenderness at the beginning of an acute attack is small and limited to the site and position of the appendix. Later on when the peritoneum becomes involved the area of tenderness is more extended. The tenderness is greater over a forming abscess or over the area of a diffusing peritonitis than over an abscess already formed, although even in the latter it may remain exquisite until relief is obtained. General, diffuse or diffusing peritonitis is characterized by extreme general abdominal tenderness. The sudden subsidence of tenderness should be taken significantly as evidence in connection of gangrene of the appendix. This subsidence of tenderness, particularly shortly following an abrupt onset in which the symptoms had been distinctive, is a bright red beacon light of danger.

The next important symptom is the rigidity of the abdominal muscles. This rigidity may be situated only over Poupart's ligament and may be more noticeable in a fan-shaped area, the center being the anterior superior spine of the ileum on that side, and may be in the flank, or may comprise the entire abdomen. In the beginning of this rigidity the patient seems to have a certain amount of control over it, but later on when the pain becomes more localized in the iliac fossa the abdominal muscles on the right side present a constant, often board-like resistance to the palpating hand. When considerable pus formation has taken place, and particularly when the pus formation occupies the pelvis, the rigidity may be both of the lower recti and the lower portion of the abdominal wall.

The next important symptom is nausea and vomiting, which is observed almost constantly in acute appendicitis. The vomiting may occur only at the onset of the attack which is followed by nausea which is continuous. As a rule the vomited material consists, first of the gastric contents, then of bile or bile-stained fluid, and finally, if septic peritonitis supervenes, of stercoraceous material.

The last of the cardinal symptoms is leucocytosis, present in the majority of cases of appendicitis. If the patient is reacting well to the infection, the white blood count will be high. If on the other hand, the system is overwhelmed by the infection, the number of leucocytes may not be increased much or be none above normal, and this always indicates great danger. A moderate count of from ten to fifteen thousand is the usual complement of an ordinary catarrhal or interstitial appendicitis, while higher counts indicate more active changes or a rapidly developing peritoneal involvement.

Constipation is the rule in the majority of cases. Diarrhea is sometimes present at the onset, usually associated with intense pain and a less favorable prognosis. Neither constipation nor diarrhea is diagnostic of the condition.

The temperature varies greatly in different cases of the same nature and in general is a most unreliable factor in both diagnosis and prognosis. Commonly at the commencement of an attack there is a fever that mounts to 101° to 103° F. or more. The temperature cannot then be depended upon in the way of judging the course of the condition, because ofttimes in spite of the development of severe complications, the temperature drops to normal. The fact of the sudden fall of the temperature to normal or subnormal must not be looked upon as a favorable sign. Ofttimes it is a sign of the rapid progress of gangrene or the rupture of an appendix or a peri-appendicular abscess. The pulse rate corresponds to the tempera-

ture, and when after an increase (corresponding to the temperature elevation) of pulse rate there is a sudden drop, to perhaps even a slower than normal rate, consideration should be given to the presence of gangrene or a rupture of the appendix or a peri-appendicular abscess.

Chills are among the rarer manifestations of general infection in appendicitis. Their occurrence at the onset of an attack, particularly when accompanied by an initial high temperature is almost pathognomonic of gangrene of the appendix. Later on they may indicate the presence of some complication of a septic character, but care must be taken to differentiate them from the purely neurotic manifestations in some patients. The development of peri-appendicular abscess is not accompanied by chills, because the abscess is walled off.

Icterus either before or after operation, when not directly due to a liver complication is a manifestation of severe and general toxemia. Respiration may be affected in appendicitis either because of a voluntary inaction of the abdominal muscles or by the presence of marked tympanites or general peritonitis, in which we find the breathing almost entirely costal. There may be some changes in the urine which is usually diminished in amount, of increased specific gravity, dark in color, and often contains albumin, casts and increased amount of urobilin and indican. The general condition of the patient varies in different instances. In the early hours of an attack there is usually an expression of pain and considerable anxiety. The patient usually lies on the back from preference, favoring the right side, the characteristic pose being that in which the body is somewhat inclined to the right, with a slight flexion of the right thigh while there is a normal extension of the left. As a rule the patient requests to be kept perfectly quiet, does not desire to be moved because of provoking or increasing the intensity of the pain. As the lesion becomes more widespread and a peri-appendicular abscess or general peritonitis develops, the expression becomes more anxious—facies abdominalis.

On examination of the abdomen there may be revealed more or less bulging of the right iliac fossa, but this in my experience is rarely present early in the case. When it is present it is due to a peri-appendicular abscess, muscular rigidity or to a tympanitic distention of adherent intestines. The general abdomen may be distended, even in the early stage of the disease, due to paralysis of the intestine because of the infection, constipation, accumulation of gases, or sometimes the excessive use of opium. Most cases, however, have no distention, and when it occurs distinctly it means the development of serious complications. On palpation we are usually able to detect

more or less tenderness in the right iliac fossa with rigidity. In distinct cases nothing more than these can be made out. My advice here is to exercise more care than is usually done in finding small areas of rigidity in the muscle planes. It is not uncommon even in quite severe pathological involvement of the appendix, especially acute exacerbations of chronic appendicular conditions, that the accompanying rigidity is not marked, but covers only a very limited area, this area perhaps somewhat removed from the anatomical site of the appendix itself. When tumor is palpable it is generally smooth or roundish in contour, its edges sloping, and means the presence of an abscess. This tumor in the vast majority of cases is immobile. Generally it is firm, but it may be quite soft, and in some instances, particularly if it be large, it may present distinct fluctuation. The presence of this tumor or swelling depends upon a number of factors. Sometimes there is practically no peri-appendicular suppuration and it is due to the thickening and edema of the inflamed tissues, to peri-appendicular serous, sero-fibrinous, and fibrinous exudate, to inflammatory alterations, not only of the viscera, but of the parietal peritoneum of the iliac fossa and the abdominal wall, and in some instances to serous and cellular infiltration of the transversalis fascia and the abdominal muscles. I believe with Deaver that it must not be assumed that all tumors, even if they be of moderate size, have within them a purulent focus. With some of the largest masses I have felt in cases of acute appendicitis it was not possible to demonstrate the presence of pus, even a microscopic quantity. In the course of time, however, such large masses usually become purulent since the process is an infective one, and then pus would be met with.

SYMPTOMS OF APPENDICITIS IN INFANCY AND CHILDHOOD.

Appendicitis in infants is very difficult to diagnose as the symptoms vary greatly from those in older children and adults. The almost complete absence of subjective symptoms makes the diagnosis almost impossible. Pain and tenderness are difficult to elicit and more difficult to localize. Vomiting, however, occurs definitely, although on the other hand it is so frequent in gastro-intestinal, nutritional and toxic conditions of infants that it is not of much value. Muscle spasm or rigidity of the right rectus may be present, but it is difficult to elicit. Manifest chill which is rare in children is sometimes noticed early. It must be remembered here that convulsions take the place of chills in the infant, and usually follow a temperature.

Constipation is the rule. The blood examination is very important in children because it usually presents a polymorphonuclear leucocytosis. The second most invaluable point is rectal examination. Having an infant with a temperature which without definite cause persists, particularly when there have been gastro-intestinal symptoms, it should be remembered that acute appendicitis is possible, and that when undiagnosed and unoperated upon, the rate of mortality is high, thus a leucocyte count and a rectal examination of the appendix should always be made. In older children the rapid progress of gangrene and perforation is much more common before the fifth year than later in life. In Porter's patients of five years or under perforation occurred in seventy-three per cent. in contrast to 28 per cent. of the ages between ten and fifteen. The greatest danger to these little patients lies in the fact that the parents are allowed to think that stomachache is a normal event in children's lives, and to believe that purgation is a panacea. It should always be kept in mind that in infancy and childhood almost every acute appendicular inflammation is rapidly complicated by an abscess.

SYMPTOMS OF CHRONIC APPENDICITIS IN THE ADULT.

Chronic appendicitis is the most common of all abdominal conditions. The diagnosis of chronic appendicitis is not difficult, yet it is the condition in the abdomen in which the greatest number of mistakes are made. This is due to a number of reasons. The average medical man, and even the surgeon, commonly makes a diagnosis of chronic appendicitis on simply the presence of a tenderness on pressure in the right iliac fossa, too little attention is paid to the history of the case because the operative mortality in chronic appendicitis is so low that many men are willing to take the chance of making a mistake in the hope that benefit will be given to the patient, and the fact that the appendix is not an important organ and that individuals can live without it quite well. Added to these is the frequency with which diseased appendices are met with, in my observation in a large city hospital, in incidental appendectomies—that is appendices removed during the process of operation upon other organs of the abdomen—in about 40 per cent. of all individuals over 35 years of age.

In a broad way it may be stated that clinically, chronic appendicitis may be classed in three divisions, relapsing appendicitis, recurring appendicitis and chronic appendicitis with referred symptoms. Relapsing appendicitis is one in which an appendicular inflammation

is always present but only shows itself as an acute lesion after certain intervals of comparative quiescence. The diagnosis is not difficult, because we have the history of one or more acute attacks, with a continued tenderness over the appendix and perhaps some digestive disturbance which goes under the term of appendicular dyspepsia. The recurrent form is one in which the acute attacks are said to be separated by periods of perfect health. It probably is doubtful whether an acute or even a subacute attack of appendicitis ever runs its course to recovery leaving a perfectly healthy appendix, pathologically considered. It may be that the feasibility is complete as far as the subjective symptoms of the patient are concerned, but on careful examination even here it is generally possible to make a diagnosis of chronic appendicular trouble. Appendicitis with referred symptoms is somewhat more difficult of diagnosis. That chronic appendicitis may give no localizing symptoms or signs and yet cause distressing "indigestion" may now be regarded as positive. Many of these cases are met with in operation for gastro-duodenal ulcer, gall-stones, adhesions or other conditions within the abdomen, and the confusing point is that most of the referred symptoms are in the upper abdomen while the appendix occupies the lower. It may here be mentioned that the type of appendicular disease described by Dieulafoy, namely, toxic appendicitis in which hematemesis is present in the individual, is one of the most confusing types. Fortunately this type of hemorrhagic gastritis, due to toxic reasons from an appendix, are not commonly met with, but in the presence of a hematemesis in an individual the possibility of chronic appendicular disease being its cause should always be kept in mind. The diagnosis of chronic appendicitis may be offered under the following caption, it being remembered that a combination of more or less is present in most cases.

HISTORY.

Leaving appendicitis in infancy and childhood out of consideration, history is very important. Those cases which give a history of recurring pains in the right iliac fossa, the attacks of pain lasting for from several hours to several days, or pain situated in the median aspect of the abdomen, there being no assignable cause, are always suspicious. The next suggestive type of history is that of an individual who has had for a length of time attacks of cramps in the epigastric region. While many times these attacks are due to gall-bladder pathology, or probably pathology in the large intestine such as a chronic colitis, it must always be kept in mind that a chronic appen-

PLATE XXVIII

Characteristic pylorospasm and hypermotility (cramps) in chronic appendicitis. (X-ray by author.)

dicitis has a distinct history of cramps independent of meals, character and amounts of food, these cramps being situated in what might be termed the stomach region, usually in the median line. The next most important type of history is the one in which no assignable cause can be ascribed, where a continued history of gastric distress characterized by a gastric hyperesthesia and hyperacidity exists. Perhaps on test-meal a distinct hyperacidity can be diagnosticated, yet the symptoms are distinctly those of distress coming on from a half to an hour and a half after eating, accompanied by gas, and considerable relief on the taking of food. This type of history is not uncommonly found in gall-bladder pathology but it is characteristic also of chronic appendicular disease—in ulcer there is usually distinct pain, not only distress.

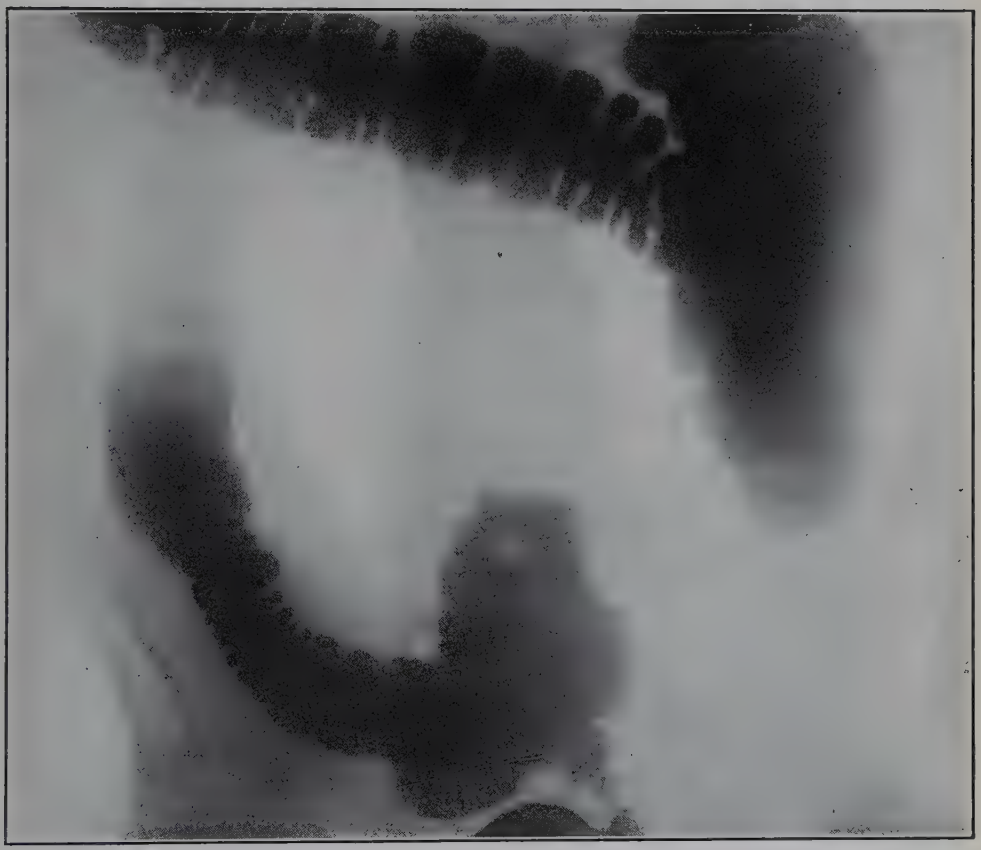
Tenderness on Pressure.—While the teachings of McBurney were important in drawing the attention of the profession to a spot of localized tenderness situated midway between the anterior superior spine and the umbilicus in diagnosticating appendicitis, it is no longer to be depended upon. It must be remembered that the base of the appendix is usually situated at an identical point in the cecum in most individuals, but that the cecum may be situated anywhere from a rather high level in the iliac fossa to perhaps well over the brim of the pelvis, even as far as the caput being in the median line. With this change, which represents practically a curved line of five inches in length, the appendix in different individuals is situated anywhere between the two ends. Therefore it is, that if one takes a point midway between the anterior superior spine and the umbilicus, exerts pressure upon it and elicits no tenderness, it may be absolutely misleading. It has been my custom and teaching for years that the first thing to do is to properly percuss out the limits of the cecum, which in the prone position is generally high enough above Poupart's ligament to be easily percussible. This is true because the cecum generally contains considerable gas, and if one approaches the cecum from Poupart's ligament, from the median line of the abdomen about midway between the symphysis pubis and the umbilicus percussing on toward the cecum, and then percussing from the flank on to the cecum, making marks at the percussible margin, it is very easy to map out the cecum for a distance of several inches in most every adult human being. If one will then recall the anatomy, that the base of the appendix begins at a point about an inch and a half from the lower pole and about an inch from the inner aspect, pressure at that point would approximately represent the base of the appendix, and distinct tenderness here means that the appendix is most probably

diseased. It has been my observation that while in chronic appendicitis the course of the appendix beyond the base usually represents more pathological disease than is met with at the base, nevertheless there is a tenderness in such appendices at the base and thus diagnosis is comparatively easy. And then too if the base is tender from pathology, the rule is that more pathology exists toward the tip.

The next step to this, after having mapped out the cecum and marked out the site of the appendix, is to make pressure directly downward, and undertake by keeping the tips of the fingers outside of the outer edge of the right rectus muscle to pinch the appendix against the iliacus muscle. In cases of chronic appendicitis there is usually a distinct tenderness which might not be elicited on direct pressure downward, but which becomes distinctly manifest on pinching it against this muscle. My observation has been that in cases of pathological disease of the cecum, this tenderness is distinct, is diffuse, and occupies more or less of the site of the cecum toward the flank outward. In cases of Lane's kink when there is an inflammatory thickening usually the area of tenderness is well inside of the percussible margin of the cecum. In cases of ovarian diseases on the right side, generally the tenderness is well below the lower pole of the cecum.

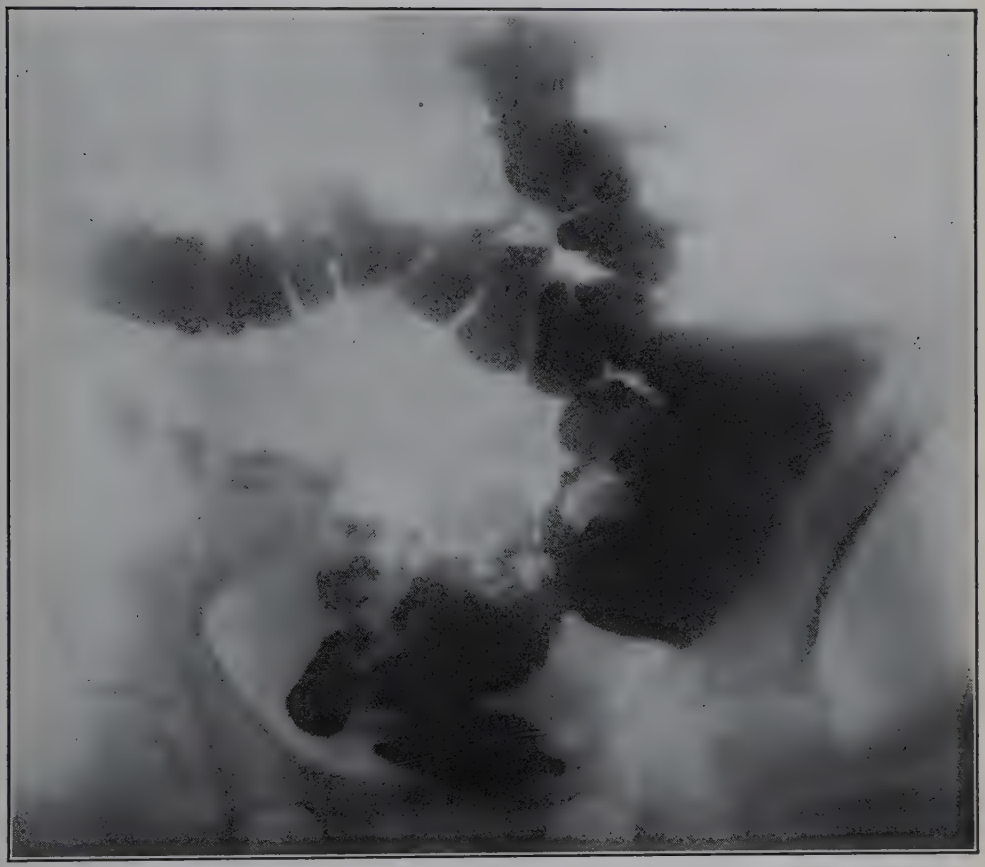
The next sign of value is that described by Meltzer in which after percussing the position for the base of the appendix, the patient being prone, a steady pressure downward is made, the patient being asked to raise the right leg at the same time. In doing so there is a pinching of the appendix against the psoas muscle on that side, and in the presence of distinct appendicular disease the limb cannot be raised without more pain than the patient can stand.

Three more symptoms may be described together. They are, first, a tenderness in the lumbar plexus on the appendix side, not on the left. This was described by Morris, and is present in many instances of chronic appendicitis. The lumbar plexus is situated about an inch and a half out and about three-quarters of an inch below the umbilicus, direct and deep pressure downward being important to bring out any tenderness. It is a difficult thing to do with a thick abdomen but is always worthy of a trial. According to Morris, if a tenderness exists in the left lumbar plexus as well as in the right, disease in the pelvis is suggested instead. The second is the presence of a hyperesthesia in the skin situated over the cecum supposed to be referred from a diseased appendix. In my experience this is not to be depended upon, and it is very difficult to elucidate because when the symptoms already pointed out have been gained in the

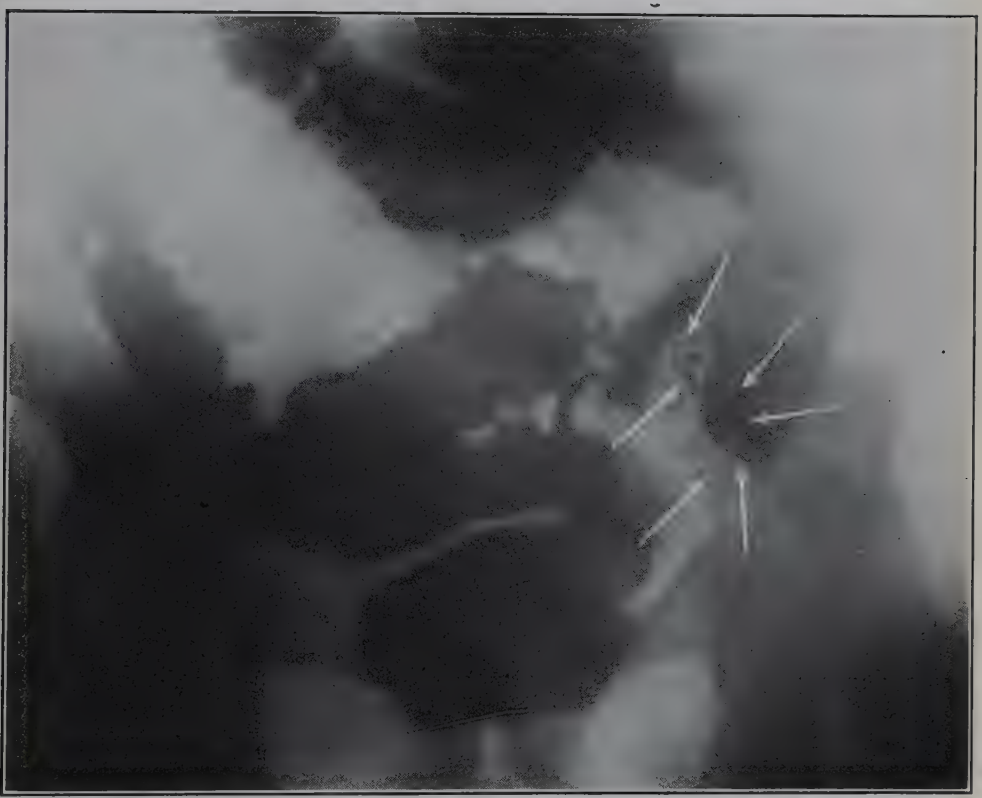


X-ray of colon, showing chronically diseased and adherent appendix.
(X-ray by author.)

PLATE XXX

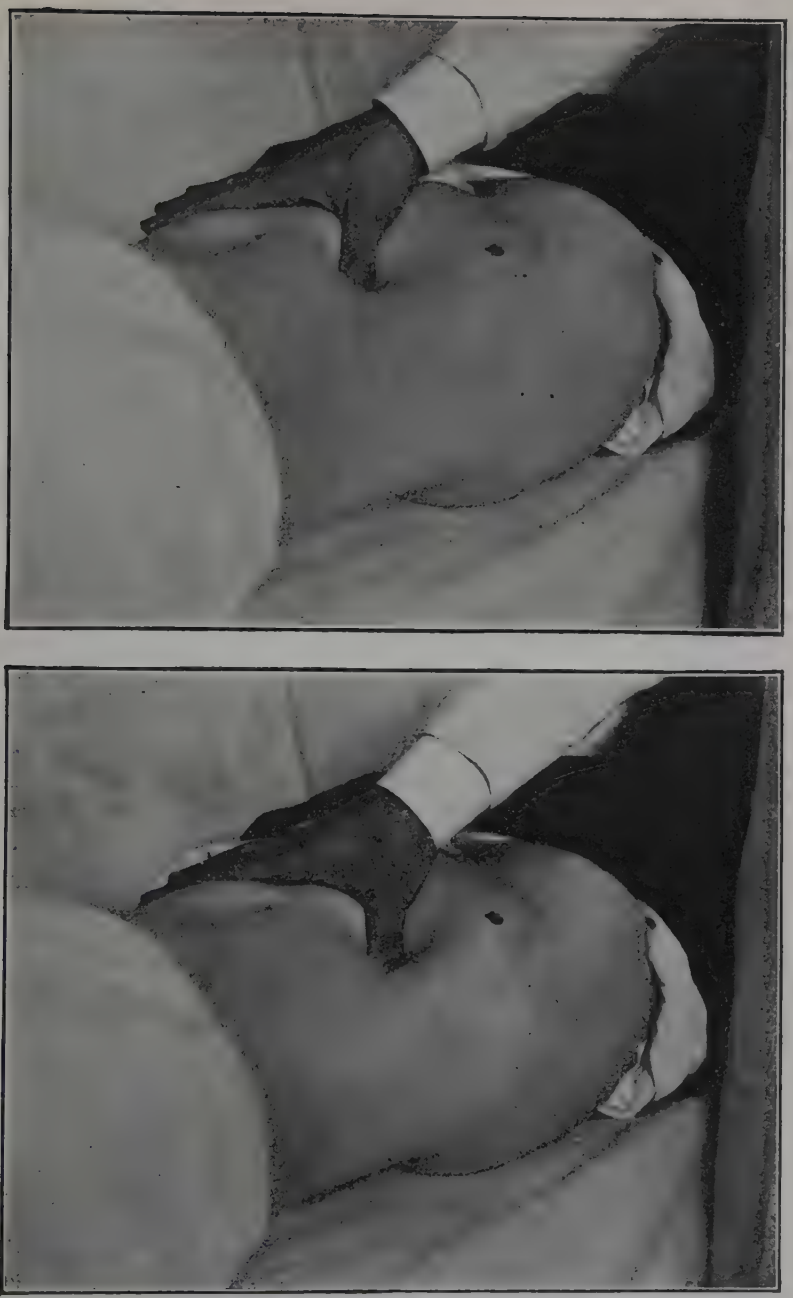


Periappendicular adhesions following chronic appendicitis. Adhesions binding down appendix region and reaching to hepatic flexure, causing constriction of transverse. (X-ray by author.)



Chronically diseased appendix, kinked at inner side of cecum. Post-cecal most of its course. Tip hanging below pole of cecum. (X-ray by author.)

Vertical appendix. Tip occluded by chronic disease. (X-ray by author.)



A

B

Fig. 80.—Pinching the appendix. *A*, Shows pressure on a line midway between the umbilicus and the anterior superior spine of the ileum on the right side, the latter marked with a black dot. *B*, Swinging of the thumb to the right of the patient, and pinching the appendix against the iliacus muscle. Patient viewed down the right side, head to left of the photograph.

case, the patient's mind and attention are directed to that side and they will claim that they have more distinct sensation on the right than on the left. The third symptom is the so-called Bastedo sign in which air is pumped into the rectum by means of an ordinary atomizer bulb, the colon is distended with air, and in the presence of a markedly diseased appendix there is supposed to be an appreciation of the pain on the right side. After studying this symptom for a number of years I have found that it has been present in cases of normal appendices and not present in instances of true chronic appendicitis. Perhaps in 50 per cent. of cases it is positive.

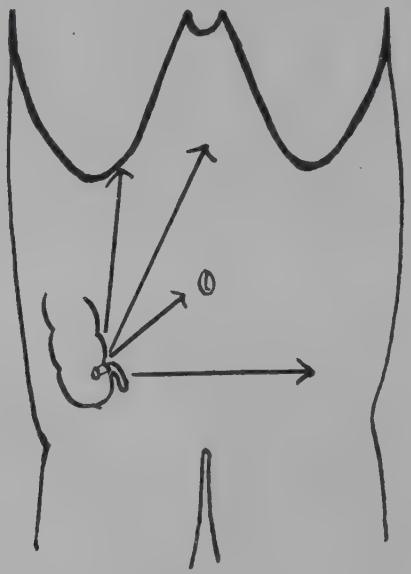


Fig. 81.—Pressure upon the appendix, referred symptoms in chronic appendicitis.

In instances of women with thin abdomens it is sometimes possible to do a bi-manual examination, having the outside hand at the proper anatomical point representing the appendix region, the other hand with the fingers in the vagina pressing upward on the right side. While it is confusing in cases of distinct disease of the right ovary to separate an appendix from an ovary by this examination, nevertheless it is one of the means together with those mentioned that may be helpful.

In a large experience of abdominal diagnosis it is often possible by physical examination to intensify one or more of the three appendicular reflexes met with in cases of chronic appendicitis. The first of

these is the epigastric reflex wherein pressure upon the appendix causes a sensation of distress in the epigastric region. This sensation of distress may not be referred as high as the epigastric region, it may be described as just above the umbilicus or in the umbilical region, but when present it usually is distinct, and not uncommonly it is accompanied by a sense of nausea. The next most common reflex is a sense of distress in what may be described as the gall-bladder region by a pressure upon the appendix, and the last is a sense of distress on the left side of the abdomen occupying about the same position as the appendix does on the right.

The Roentgen examination of the appendix since Holzknect's original studies in 1907 has made rapid advance. Diagnosis here rests upon the possibility of being able to fill the appendix with barium. A study of the course and the diameter of the lumen of the appendix throughout its course, movability or fixation by fluoroscopic examination, and the length of time that it takes the appendix to empty are all important. One of the confusing points here is that the appendix may be considerably diseased, particularly at its base, and thus no barium gain entrance into it, in which instance it is not visualized, or again it may be post-cecally situated and thus not show in the plates which demonstrate a filled right side of the colon, the appendix only visualized after all the bismuth in the colon has disappeared, and even then the appendix may be diseased not permitting any bismuth to gain entrance into it and not be visualized at all. In the routine of the X-ray work as I go along, I visualize the appendix in at least half of all the colons examined. It is important in this connection that the patient be examined prone, and preferably by fluoroscopic examination at the time that the clysma is given, allowing sufficient time for it to reach the ileocecal region. Not uncommonly the appendix can be visualized directly, or it may be possible to roll the cecum into the flank, exposing more or less of it in this way. By this examination it is possible to make direct pressure upon the appendix and elicit the presence of tenderness on pressure, this method of course being very accurate from an anatomical standpoint. When the appendix can be seen filled, Roentgenographic plates should be made and a study of its shape and size, a fluoroscopic observation being necessary for a study of its movability and tenderness. The presence of concretions and kinks, adhesions, the question of drainage, the relation of the visible appendix shadow to the point of pain on pressure, the position of the appendix whether retrocecal, post-cecal, etc., all are easily possible. Often the lumen is seen to be constricted and important conclusions may be

drawn from a broad appendix shadow persisting for a long time, perhaps for several days, after the rest of the bowel has been empty. The exact position of the appendix is often suggestive of a pericolonic membrane of a variety described by Eastman. Kinks of the appendix are frequently met with under the fluorescent screen examination. It is my custom to examine the appendix region about eight hours after the bismuth has been taken by mouth and before the clysma is given. In many instances sufficient appendix shadow can then be made out, although care must be taken that it is not confused with a thin line shadow of the lower ileum commonly seen.

A non-functioning appendix may be incapable of receiving material because of the obliteration of its canal. It may receive it and only discharge part, retaining the residue for an indefinite period. I have observed appendices which retained bismuth for as long as two weeks, and it may definitely be stated that the non-functioning appendix is always pathological, either having changes within its walls or dense adhesions surrounding it. A fixed appendix is usually adherent to the abdominal wall, otherwise it is attached to the adjacent viscera that are held by adhesions which prevent displacement by manipulation. It may be that only a portion of the appendix is fixed, and if so it usually is the tip or the end. As a rule the ascending type is adherent, the tip may extend to the transverse colon or the liver. Descending appendices are most often found to be normal.

After considerable experience with the Roentgenographic method of diagnosis of chronic appendicitis I have come to the belief that it is not a safe method to be depended upon alone. The history or physical examination, preferably both together, are essential for accuracy in the work. There are many other abdominal conditions, and the instances of diseased appendices in individuals of middle age are found in almost half. When a case comes under observation for abdominal distress in which it is judged that some organic condition is present, and when this abdominal condition is one of appendicitis, the diagnosis is comparatively easy, either from the history alone or from the physical examination. But when, on the other hand, these are not distinctive, and one's attention is too much directed to finding diseased appendices and the work is entirely an X-ray diagnosis, mistakes in what is really the cardinal disorder with the particular individual are liable to occur. It is true that when a diseased appendix is present its removal may be justified, but what patients come to us for is a diagnosis of cause of the abdominal distress, and it is not uncommon when a diseased appendix is present that a promise is made of the relief of the symptoms if the appendix is removed, but

when it is removed none or but very little benefit is brought about in the case. Many of the mistakes that have been made in this way have been made by those who depended upon the X-ray diagnosis alone, and, while I am partial to this means, at the same time I would suggest that instead of it being used as a primary method of diagnosis, it be used as far as possible as a confirmatory means. Only in this way can one's diagnosis be as broad as is necessary for accuracy. In my clinical and hospital experience many are the instances in which a diseased appendix must be removed for the subsidence of symptoms in the case. There are many others having symptoms in which perhaps the X-ray examination proves the presence of a diseased appendix but in which if only the appendix is removed the symptoms would go on as before. When we are called upon to diagnose the cause of symptoms in the abdomen we must always keep in mind that in the abdominal cavity there are more organs than the appendix that can become diseased, and that the removal of even a diseased appendix cannot cure every one of them.

TYPHOID APPENDICITIS.

Symptoms.—Typhoid appendicitis may occur during the course of typhoid fever, or typhoid fever might light up an intercurrent appendicitis and it is probable that appendicitis in a primary way can come from the Bacillus typhosus.

During the course of an attack of enteric fever most of the lymphoid structures of the lower ileum and cecum have infiltration of the lymphoid tissue in which the appendix may be involved. This is true in about two-thirds of all cases of typhoid fever. This being so, it must be plain that in the majority of typhoidal infections wherein the appendix tissue is also involved, there follows a complete recovery at the subsidence of the primary disease. There are cases, however, in which during the course of typhoid fever the added infection of the Bacillus typhosus to one already present in the appendix causes the establishment of an acute appendicitis which may require surgery. It is well known that in typhoid fever perforation of the appendix may occur, according to Finney, representing 5 per cent. of all the typhoidal perforation cases. Green has called attention to the fact that typhoid fever may arouse a latent appendicitis, bringing it into prominence and making operation necessary. Then there is a third group of cases of primary appendicitis due to typhoidal infection in which there are no other lesions of typhoid fever. These are very few in number and it is a question whether taking the many

more cases in which typhoid fever arouses a latent appendicitis that the typhoid infection is merely incidental or rather an exciting cause to an already chronically diseased organ.

In instances of intestinal perforation in typhoid fever it must be kept in mind that the appendix might be the organ in which the perforation occurs. Such cases must also be differentiated from intestinal hemorrhage, thrombosis of the iliac or femoral veins, and affections of the gall-bladder, all of which are comparatively easy.

Differential Diagnosis.—While in the majority of cases the recognition of appendicitis is not difficult there are nevertheless some affections that in many ways so closely simulate it as to give rise to perplexities. Of these the following may be mentioned:

Gastro-intestinal inflammations represent a class of cases of which such diagnoses as acute gastritis, acute gastro-duodenal catarrh, acute enteritis, simple intestinal colic, dysentery and colitis are divisions. As a rule there is a history of the ingestion of some food, indigestible either because of its quantity or quality, followed by nausea, vomiting, flatulence, colic, etc. The history is a very important guide in differential diagnosis, although too much stress must not be laid upon it because very often during an attack of what appears to be acute indigestion the appendix is primarily at fault.

Careful physical examination must be made in each case because the initial pain of acute gastritis and enterocolitis very much resemble that in some cases of appendicitis. The tenderness is usually less constant in the conditions noted, and none of them are sharply localized in the right iliac fossa. Acute gastritis may give rise to some temperature which is liable to be somewhat confusing, but here we generally have the history of the taking of indigestible foods, conditions in which the stomach is relieved when emptied, and perhaps the presence of cardiac diseases as a predisposing factor. A case of acute gastroduodenal catarrh shows itself by symptoms similar to those of acute enteritis, with perhaps the addition of a slight icteric coloring of the skin and sclera. There may be some rigidity for a time which is generally bilateral, localized to the upper abdomen and quite resistent, the right iliac fossa being essentially negative. The same may be said about intestinal colic or enteralgia, wherein appendicitis may be excluded by examination. In acute enteritis in the adult differentiation is easy. In children it may be very hard to recognize it from appendicitis with diarrhea, because a history may not be obtained, and because in children diarrhea is far more common in appendicitis than in adults. A painstaking physical examination will however, if repeated and made when the voluntary spasm

of the abdominal muscles is in abeyance, give us some clue to the real nature of the disease. Dysentery and colitis are merely special forms of enteritis, in which there is usually a distinct colic with intermission, not as sharp and constant as that of appendicitis. There is relief upon pressure of the abdomen, the patient being quite restless and distressed like those with attacks of acute colic. The establishment of enteritis distention and borborygmi are features which lead to the diagnosis of a catarrhal condition of the small or large intestine. In the gastro-enterocolitis group the rigidity is largely a voluntary matter and may be overcome by patience, whereas in appendicitis the muscular guard is unremitting. In chronic intestinal stasis there should be no confusion because there are but few symptoms in an abdomen as a rule, these being comprised mostly in such symptoms as indefinite distress, gas, perhaps some distention of the right side, while the general condition of the patient is one making it easily possible to make a diagnosis of intestinal toxemia. In the cases of intestinal stasis not uncommonly there is considerable distress in the cecal region due to some disturbance with the normal flow of fecal content, probably also accompanied by gas. Such cases have been variously diagnosed as pseudo-appendicitis, ileocecal pain, and even typhlitis. In my experience, when one is careful with the history and examination, differentiation is not difficult.

In chronic gastric and duodenal ulcer if one is careful in the history and the physical examination, particularly if the X-ray has been added, differentiation is not difficult. In not a few of these cases the appendix is also diseased. I have seen several cases in which some symptoms were distinctly due to the ulcer on the one hand and to a chronically diseased appendix on the other. The presence of perforation of gastro-intestinal ulcers gives rise to symptoms which closely simulate those of a sharp acute appendicitis. But the distinction between the two types of cases is not difficult. The same may be said of cases of intestinal obstruction with appendicitis. There are a few cases of rapidly fulminating perforations of the appendix followed by a spreading peritonitis in which quick paresis of the bowels and obstipation is so marked that the case may look like one of mechanical obstruction. However, the history and the examination of the appendix region, the prominence of vomiting, and the rigidity and tenderness being rather more general than represented in the case of peritonitis serve as distinguishing features.

Carcinoma may be primary in the appendix and is rather hard of diagnosis without operation. Usually the history shows a gradual onset of the condition, it taking weeks before the symptoms are dis-

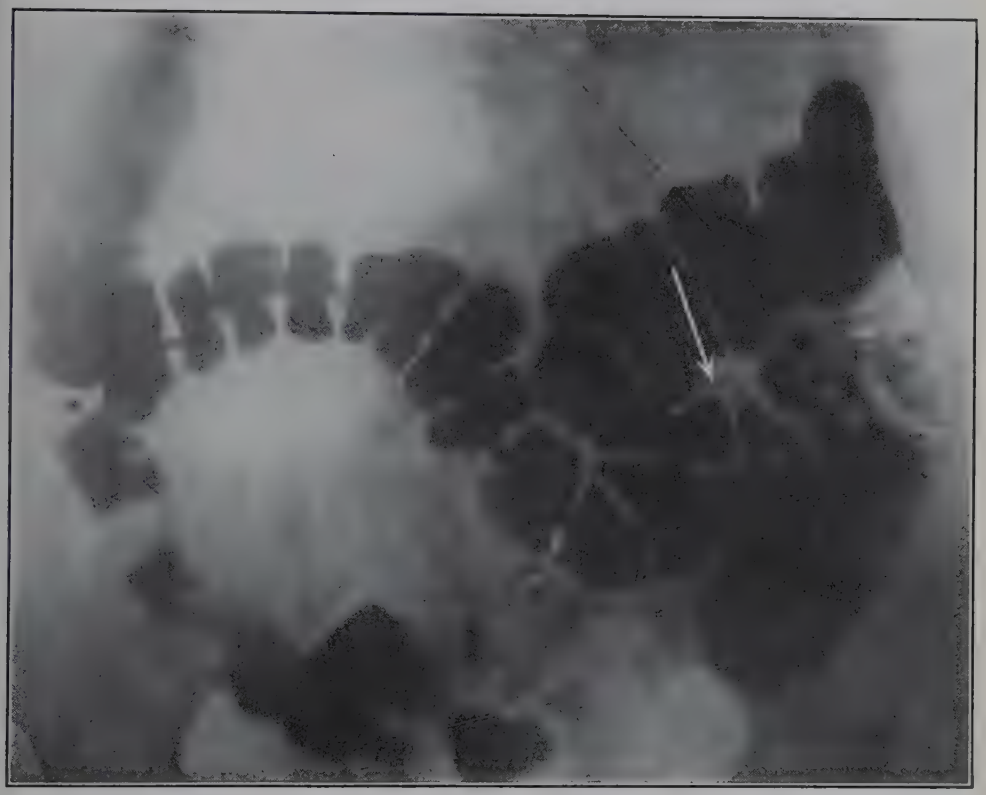
tinctly present and, in the absence of inflammatory symptoms, when the blood examination shows an anemia, there being an alternating constipation and diarrhea with an occasional track of blood in the stools present differentiation is not difficult. The same may be said of diverticulitis, which in my experience in cases wherein there has been the development of a perienteric abscess, they in every instance have been on the left side, usually quite deep in the pelvis. An ulcer of the ascending colon may also give great difficulty, particularly when that ulcer has perforated—in this instance a malignant disease is usually associated with it.

It may be mentioned further that cases of acute appendicitis, and even the chronic forms, have been mistaken for various diseases of the gall-bladder and biliary duct, empyema and stones of the gall-bladder particularly. While it is believed by surgeons that differentiation between some cases of appendicitis and cholelithiasis is far from an easy matter, it has not been so in my experience. Chronic appendicitis is far more liable to give as a referred symptom in the epigastrium a cramp-like character than is disease of the gall-bladder. It is not uncommon in disease of the gall-bladder for symptoms of gastric distress to come on an hour to two hours after meals, and usually it is not present or is much slighter in cases of appendicitis alone. An examination of the gall-bladder region, particularly with the patient sitting, being sure that the examining fingers are at the median fissure of the liver, eliciting a distinct tenderness there that is not present on examination of the appendix with the patient prone, is most helpful in differentiation. In gall-bladder conditions, cholelithiasis particularly, the referred symptoms are usually in the right side of the upper abdomen and chest, there may be a tenderness along the lower intercostal basin, pain is referred to the right shoulder caused by an irritation of the phrenic and commonly one gets a history of colic in the biliary location, especially notable in women who have had children. One should have no difficulty in differentiating appendicitis from cases of hepatic or perihepatic abscesses or chronic pancreatitis, some kidney conditions, diseases of the ureter, extra-uterine pregnancy, diseases of the female pelvic organs, pneumonia and pleuritis.

Prognosis.—Little can be stated regarding the prognosis in appendicitis because of the vast variety that exists in the cases, and the estimation of prognosis would have to be based upon definite pathology which is impossible. Thus with this complex of acute and chronic pathologies any remarks on prognosis would be quite useless in a practical way.

PLATE XXXIII

Kinked appendix, retro-cecal and chronically diseased. This observation was made on fourth day following barium by mouth. (From author's service in the Polyclinic Hospital.)



Appendix chronically diseased. Tip adherent to transverse colon. Symptoms, appendicular dyspepsia. (X-ray by author.)

PLATE XXXV

Vertical appendix. Tip adherent to the ileum, base low because of prolapse of the cecum. Eight-hour observation. (X-ray by author.)

Injected colon in a case of left sided appendicitis. (*Case.*)

Treatment.—The treatment of appendicitis, whether acute or chronic, is operation as soon as possible after the diagnosis is made. It is an insult to American medicine and particularly to American surgery, to present what might be termed medical treatment for appendicitis. All that is necessary for a physician is to have considerable experience in a large city hospital for him to come definitely to this conclusion. To write or teach otherwise is like taking the reader out of the light and putting him in the dark. Not uncommonly some of my students ask the question whether it is not wiser to treat a patient during an acute attack and operate in the interval. In my opinion there is no interval. Once an appendix is diseased, whether the symptoms are acute or chronic, operation is in order. One reads in the newspapers of people dying of acute appendicitis, this taking place at the time or shortly after an operation. After considerable consultation experience it has been my observation that often these are cases which were treated medically, with the idea of operating in the interval, but the pathology, being such that allowed no interval, killed the patient instead—namely the operation had not taken place soon enough to prevent a peritonitis. There are a few instances in my experience where there has been one attack of appendicitis, and never a return of the symptoms. I can count these on the fingers of my two hands, while each finger has been manifolded by the score of instances of cases treated medically and operated upon too late. Thus I have come to the conclusion that whatever the character of the clinical aspect, whether it be that of acute appendicitis, chronic appendicitis, exacerbations of chronic appendicitis, or however it may seem at diagnosis, operation is in order, and operation as soon as preparation therefor can be made.

Accepting that as the dictum of the wisest course to pursue in all cases of appendicular inflammation, there may be added what to do in instances where acute attacks have taken place and operation cannot be resorted to at once. After the diagnosis is definite, and arrangements have been made for the operation, the patient can be comforted by a withdrawal of food, an ice bag or hot application placed on the right side of the abdomen (whichever feels the more grateful), complete rest, and perhaps the use of a hypodermic dose of morphine. I request that my mention of morphine be not misconstrued. I feel satisfied that in a few instances where I have seen cases of acute appendicitis of a fulminating type where an operation could not be performed for several hours that morphine quiets the pain and the nervous condition of the patient, and if anything, steadies that individual so that there is less shock. This is particularly true in

cases of acute perforation, gangrene, if a quick peritonitis takes place, or the operation is long delayed. The use of purgatives, either by mouth or by enema, in my opinion is contraindicated. In fact I am satisfied that I have seen many instances of disastrous effects from the use of calomel or castor oil during an attack of acute appendicitis. I have also seen harm done by simple saline enema for the purpose of unloading the lower bowel. It must always be remembered that fluid introduced into the rectum very rapidly gains access to the ileocecal region, and in this instance it may result in the spreading of a general peritonitis.

I desire again to state that the time to operate for appendicitis, whether acute or chronic, is promptly after the diagnosis is made, and this should be a cardinal rule because it is the only rule that can save the largest number of cases.

CHAPTER XII.

Circulatory Conditions.

INTESTINAL EMBOLISM AND THROMBOSIS.
(Mesenteric Arteries.)

THAT part of the mesentery where the vessels enter, as pointed out by Treves, is the real root of the mesentery. For the reason, however, that the mesentery is attached to the back part of the abdominal cavity for about 6 inches below this point, the term "the root of the mesentery" in practice is usually considered to include these 6 inches of attachment. This root lies obliquely in reference to the spine, and can be marked out with a fair degree of accuracy on the abdominal wall. The attachment represents an oblique line running from the left of the patient pointing to the right groin, the umbilicus being at the junction of the lower with the upper two-thirds of the line. Although the root of the mesentery measures only about 6 inches, the intestinal border of this structure measures from 30 to 60 times as much. It is obvious, therefore, that the mesentery in order that it may accommodate itself in the abdominal cavity, must be thrown into many folds, folds which are even more numerous in instances where the length of the intestine is greater than usual. These folds are, naturally, simplest near the root of the mesentery, where the diameter of that structure downward is comparatively little increased. The first one of the main folds arises from the upper part of the mesenteric root and is generally directed to the left side of the abdomen; the next one lower down, to the right; while those arising below proceed somewhat indefinitely from the lower part of the mesenteric attachment to both sides of the abdomen and pelvis. The various coils of intestine, naturally, occupy the same regions as the corresponding parts of the mesentery.

If one examine a loop of the small intestine which belongs to the uppermost third of the bowel, he will notice that it is thick and in many cases it feels even "fleshy." The valvulæ conniventes are easily palpable as the fingers pass over them. He will observe that the intestine is of large size and very vascular, being covered with numerous branching vessels. It can easily be observed when the vessels are visible that these are large, long and straight, and that they

radiate from the depths of the mesentery directly to the intestine. Without much difficulty it can be noted that these vessels arise mostly from primary arches, deep in the mesentery. A few secondary arches may be seen, but these are usually more characteristic of the mesentery opposite lower parts of the intestine.

If one now observes a loop of the bowel with its attached mesen-

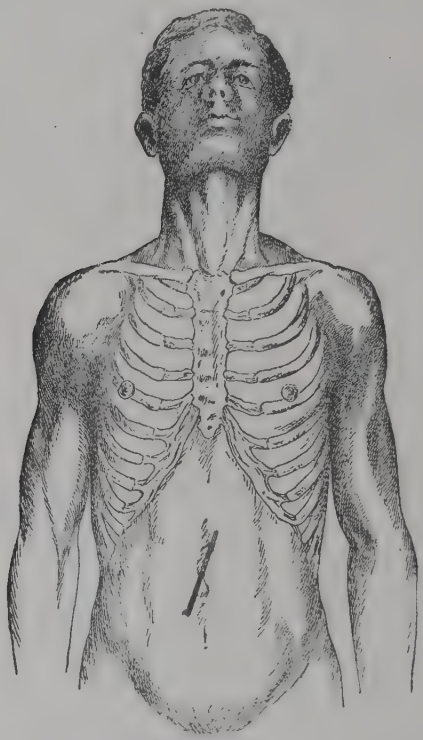


Fig. 82.—Position of the attachment of the mesentery to the posterior of the abdomen.

tery at the lower third of the intestine, quite a different picture is observed. This loop of gut is usually thin and is generally of a smaller caliber than that of the upper part of the bowel. Few valvulæ conniventes can be felt through its walls, or even none at all. Finally, it is less vascular than is the higher loop which requires an increased vascularity because of its larger physiological function. It is unlikely here that any lacteals will be observed in the mesentery, and the difference between the anatomy and the vascular supply of the two ends of the small intestine suggests very strongly that in the

upper part considerable physiological function takes place, which is not so true in the lower, and this probably explains the reason why resection of the lower part of the small intestine is distinctly more feasible than of the upper, the latter usually being fatal.

The infarcts of the small intestine may be classified into: first, hemorrhagic (*a*) through embolism of the mesenteric arteries, (*b*) through thrombosis or thrombo-phlebitis of the mesenteric vein; second, anemia, due to obliteration of the mesenteric branches below the anastomotic arches; third, curable and limited thrombi, arising through obliteration of the small or intermediate sized branches of the mesenteric or intestinal vessels. Another form has been described in

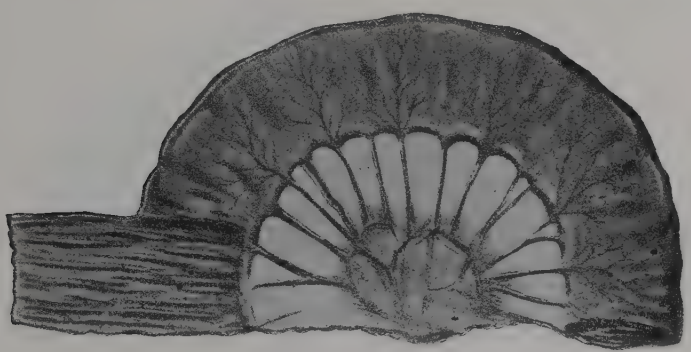


Fig. 83.—A loop with attached mesentery, from the small intestine of a thin muscular (male) subject. It belongs to the uppermost part of the bowel, as is shown by its large size, its obvious thickness, its vascularity, and by the presence of large and numerous valvulæ conniventes; and also because of the presence of large, long and straight vessels in the mesentery radiating to the gut from arches, mostly primary. There is very little fat in the mesentery, and the transparent spaces between the vessels are very extensive. (*Monks.*)

the preceding chapter under ulcers due to localized hemorrhage. It must be remembered that thrombi can occur in the hepatic, portal vein, and iliacs as well as elsewhere in the abdomen, these being commonly due to infection either as complicating conditions or as terminal results.

Symptoms.—In my experience the majority of cases of mesenteric thrombosis have not been diagnosed before operation. There are some cardinal points to be kept in mind which not uncommonly make a diagnosis possible. The diagnosis most often made is that of perforating lesion of the stomach, duodenum or gall-bladder, hemorrhagic pancreatitis, or peritonitis. One should keep in mind that cases commonly occur in persons who have other diseases such as disease of

the liver, heart, aorta, general arterial system, nephritis, or are or have recently been the subject of an acute infection. It may therefore occur in middle-aged individuals and less commonly in the old.

Usually there takes place as a first symptom a sudden, violent abdominal pain with repeated subsequent paroxysms. Hemorrhage occurs in the bowels, generally lasting for a day. Usually there is a muscular rigidity in the right hypochondrium, perhaps a sausage-like mass may be felt on palpation. There soon takes place a septic peritonitis with hiccoughs, coffee-ground vomiting, and death in a short time. The picture then would be one simulating an intestinal obstruction which may, after all, be the primary condition in cases of

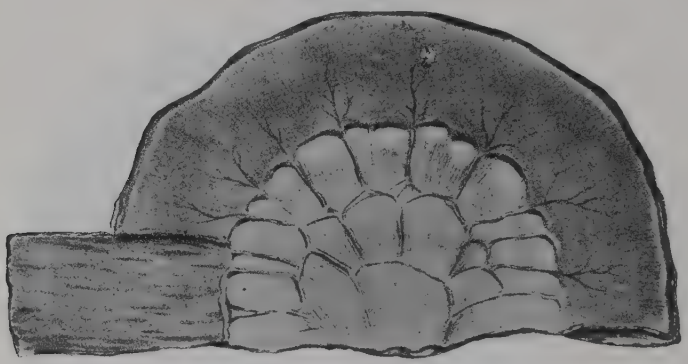


Fig. 84.—A loop, with attached mesentery, from the small intestine of the same subject (Fig. 83). It belongs to the lowest part of the bowel, as is shown by its smaller caliber, its comparative thinness, the absence of vascularity and of valvulæ conniventes; and also by the presence in the mesentery of comparatively short, small and somewhat tortuous vessels which radiate from arches, mostly secondary or tertiary. The fat in the mesentery is more abundant than in the specimen shown in Fig. 83, and it approaches nearer to the intestine. For this reason the mesentery here is much more opaque, the vessels are somewhat obscured, and the transparent areas small. (*Monks.*)

mesenteric thrombosis. It is apparent though that the picture, while striking and quick, is not particularly classical and in minor part is not unlike that seen in some cases of hemorrhagic pancreatitis, the acute diffuse form of peritonitis, or even in perforation. Moreover cases are not unusual in which the vomiting is persistent, coming on almost at the onset of the condition. The exceedingly violent pains are more severe than those met with in any other abdominal condition. We have then to remember the existence of an infection capable of causing thrombosis or embolism, and especially the accompanying hemorrhage. What is striking, however, and upon which diagnoses can often be made, is the presence of two contradictory

symptoms—diarrhea and occlusion. This combination when present is of diagnostic importance. In cases of acute pancreatitis, intestinal obstruction, perforation of the stomach, duodenum or gall-bladder, where there is a quick peritonitis from any cause, diarrhea is not a common symptom, but constipation is generally the rule. In an alarming status of illness then, diarrhea containing blood always suggests the possibility of embolic or thrombotic occlusion at once.

Prognosis and Treatment.—Attention is called to the frequency and relatively favorable prognosis of embolism of the small and mesenteric branches in contrast with the fact that in embolism and thrombosis of the large mesenteric vessels death usually results, this occurring generally about the fourth day.

In the presence of a condition like this, of course, one feels that operation should be done at once, but in those cases that I have had operated upon (some of them within a few hours after the condition had ensued) death always took place. *Post mortems* on these cases showed that although the entire bowel which had been included was removed, the condition extended beyond the point of anastomosis. Therefore it is probable that the bacteria of the intestine get out into the mesentery, produce a local lesion from which thrombosis results, first in a small area and which extends to the whole portal system. In a case of mesenteric embolism one can resect the portion of the intestine with perhaps good result. Here it may be mentioned that an embolism does not have the same leathery feeling of the mesentery that one observes in thrombosis. The condition felt in mesenteric embolism is clean cut and sharply to be distinguished from thrombosis, which, unlike it generally, continues its progress until it may involve the whole blood supply of the small intestine—and therefore it is fatal.

At the same time I do not believe with Cabot[1] that "surgery has little or no business here." The truth is that one can never tell positively whether you are dealing with an embolism or with a thrombosis, and thus the wisest procedure is immediate surgery. If it is an embolism there is possibility of recovery after resection of the intestine; if it be a thrombosis it might be wise to resect nevertheless. Death is almost inevitable in the latter, because I know of only one instance of what was distinctly a thrombosis which eventually got well. The point is that we cannot tell from the symptoms until the abdomen is opened. That being a fact, each one of these abdomens should be opened as promptly as possible. Only in that way do we give our patients the benefit of any chance at all, even though it be but little in thrombosis.

VISCERAL ARTERIOSCLEROSIS.
(Senile Dyspepsia.)

It is rather difficult to classify arteriosclerosis. Instances are met with in which the alterations are confined chiefly to the larger trunks, notably the aorta, carotids, and femorals, while all the peripheral and visceral arteries remain practically free from disease. Other cases show disease of the small or medium sized trunks with very little in the larger vessels. Then again there are instances in which the changes are microscopic, involving the smaller visceral branches. It is, therefore, impossible to describe the pathology of arterio-sclerosis in any definite way, because in almost every instance more or less combinations of the above are met with. Practically all the cases of general arteriosclerosis present disease of the visceral trunks. According to the statistics of Brooks,[2] in 400 cases of arterio-sclerosis serious disease of the visceral trunks showed in 368. Of these 400 cases 125 were women and 275 men. The statistics in which the visceral vessels were involved were about equal in both sexes, the ages of these patients running between 13 and 80 years, with an average of about 45 years.

In all excepting 30 of the 368 cases more than a single visceral distribution was found to be invaded. Disease of the coronary arteries predominated considerably in number, coronary sclerosis being found in 270 of the 368 cases. Next in frequency were the arteries of the brain of which there were 132 cases. Then came the arteries of the kidneys in which marked disease was found in 81 cases. The fourth in number were the vessels of the pancreas, which were found involved 74 times. The hepatic vessels were found diseased in 43, the splenic arteries in 35, the vessels of the lungs in 10, and disease of the visceral branches of the celiac axis was greater than the other trunks of corresponding size in 19 cases. Diffuse involvement of the mesenteric vessels was pronounced in 4 cases, all associated with adiposis. The spinal vessels appeared extensively diseased in 20 cases. In these cases the history of alcohol, syphilis, nephritis, endocarditis, tuberculosis, senility, toxic poisoning (particularly in workers in lead) and tobacco are most often met with.

Etiology.—The etiology of arteriosclerosis presents certain definite conditions in a clinical way. In careful study there is considerable suggestion that changes which are largely to appear are found first to the greatest degree in those arterial distributions which are called upon to functionate most actively under the special stimulants of work. This would explain the heart condition in alcoholism,

severe labor, or nephritis in which the increased work of the heart muscles demands a more or less heightened and prolonged physiological hyperemia of the myocardium. Similarly, in over-alimentation the same condition is demanded in the pancreatic, gastric, and hepatic vessels.

The causes in general of arteriosclerosis can be summed up under the head: 1. Toxic, that is, lead infections of all sorts; excess of internal secretions such as adrenalin, absorption of toxic material from the intestinal tract, represented in the putrefactive product; and 2. The senile or quasi-senile in which would be included the wear and tear from over-work. To these must be added tuberculosis, syphilis, and practically all the infectious diseases, in which poison chronically circulating in the blood, takes the lead, and those due to resorptive products from the intestinal canal in toxemic states—these being the most commonly met with. Habits and occupational conditions such as the excessive use of alcohol and tobacco represent a definite proportion of the cases. There is no doubt, as Metchnikoff has shown, that the rôle of intestinal putrefactive products, particularly the so-called aromatic series, is most important in producing sclerosis. In animal experimentation para-cresol or indol injected into animals caused in the course of time, definite disease of the vessels.

Pathological Anatomy.—The pathological changes which follow as a direct result of these arterial lesions differ as a matter of course according to the precise change present in the vessels, the etiological factors, and the anatomical and physiological nature of the organ affected. It can be generally stated that the first effect of the arterial disease appears in the parenchymatous tissues supplied by the vessels. These are similar to those met with in general or local malnutrition, as in starvation, old age, or local anemias. They consist, in the greater number of cases, either of an atrophy, usually with menochromatosis (brown atrophy) or of changes best classed either under the heading of fatty infiltration or degeneration. Quite naturally these are followed by functional diminution of the physiological activities of the organ, and as a logical result, in accordance with the general laws governing atrophy and interstitial growth, connective-tissue hyperplasia follows, originating from the perivascular connective tissue network or from the normal supporting stroma of the organ. In consequence of the interstitial proliferation, and especially of that in the immediate vicinity of the vessels, this newly-formed connective tissue begins to contract which is the natural developmental stage of adult connective tissue. Interstitial sclerosis with

hardening of the organ follows, and the lumina of the vessels thus becomes further compromised, leading to a still greater diminution in blood supply.

Symptoms.—The clinical picture of abdominal arteriosclerosis is characterized by abdominal tenderness and distention, by severe paroxysmal abdominal pain, obstipation, sometimes hypertension, and occasionally sudden profuse hematemesis.

Abdominal pain is a prominent symptom. Sometimes it begins with paroxysmal attacks lasting from a few minutes to half an hour, and recurring several times in a day, later on becoming continuous, the patient complaining of a dull ache and soreness in the abdomen. The pain is usually localized in the epigastric or umbilical region, less commonly in the lower abdomen. It is increased by exertion and in some instances during digestion. Instances may be noted in which the pain is brought on by emotional disturbances or the assumption of a horizontal position. Often this pain is of a darting or burning character, simulating the agony of angina pectoris, it therefore having been termed "angina abdominis." This usually occurs from two to three hours after a heavy meal, is griping and twisting in character and has been described by Schnitzler[3] and Markwald,[4] as due to intermittent claudication and ischemia following vessel spasm.

Next to pain perhaps the most common symptom is weakness, and not a few of these patients lose in weight. The loss of weight may be moderate and gradual or rapid and considerable, this loss of weight being characteristic of most all cases of advanced arteriosclerosis—but distinctly more marked in those in which the visceral vessels have been affected—namely, those that are associated with digestive disturbances. It is probable that some degree of the reduction in weight is occasionally due also to dieting which is undertaken in an effort to relieve the dyspeptic symptoms.

Abdominal distention and belching are frequently present, and sometimes the cases present a marked degree of disturbance in these ways, making one suspect, with the fullness and distress after eating, nausea and the regurgitation of sour fluid, that there is a more distinct intestinal condition such as malignant disease.

Not uncommonly patients suffer from attacks of vertigo, and a few complain of disturbances of vision. The urine is frequently diminished in quantity and may show signs of an interstitial nephritis. Stool and urine findings such as have been described in connection with intestinal toxemia are not uncommon. The radial arteries may or may not show the presence of sclerosis, although usually this is found. The most valuable method is the use of the ophthalmoscope

by means of which the retinal vessels can be directly inspected and the sclerosis noted. The blood pressure may be high, low or normal. When high it suggests a distinct involvement of the kidneys or the myocardium. In my opinion the majority of cases of distinct visceral arteriosclerosis do not show an elevation of blood pressure.

The diagnosis of arteriosclerosis can usually be made without an analysis of gastric content. When done as a routine, however, it is not infrequent that free hydrochloric acid is absent, although it must be remembered that free hydrochloric acid is commonly absent from the stomach content in perfectly normal persons over fifty years of age. The digestion is usually fair, the motility of the stomach normal, and sometimes on physical examination, a thickened, strongly pulsating abdominal aorta may be felt, particularly when the individuals are emaciated.

Obstipation is not uncommonly met with and Pal[5] explains the presence of obstipation as atony of the bowel from anemia produced by vessel spasm. Sudden profuse hematemesis simulating the hemorrhage of gastric ulcer, often with fatal outcome, is frequently the first and only evidence of abdominal vessel disease. It occurs when the sclerosis has invaded the arterioles in the stomach and upper bowel submucosa.

Diagnosis.—Diagnosis must be made between the conditions described; such as malignant disease of the stomach, abdominal crises in tabes, perforations of the stomach, duodenum and gall-bladder, acute pancreatitis and appendicitis and sometimes biliary colic, or other causes which may give acute abdominal pain. The history, general examination of the patient, the paroxysmal feature of the pain with a progressive weakness or rapidly advancing adiposis or what has been described above usually serves to differentiate the cases in a comparatively easy manner.

Treatment.—The most important factor in connection with this subject is the prophylaxis. All there is to mention in connection with the habit, occupational and toxic factors as causes enters here. The use of alcohol and tobacco should be strictly interdicted; those who work in lead or any of the other metals must change their occupation. It is probable that the benefit which has come from the use of iodine or the iodide preparations has been due to the large part syphilis has had in its production. In the administration of these remedies small doses continued over lengths of time are in order, and it may safely be said that what cannot be accomplished by one- or two- drop doses of tincture of iodine three times a day, or three- to five- grain (0.20 to 0.30 gram) doses of any of the iodides, cannot be accomplished with

iodine at all even in still larger doses. In my opinion, whatever the cause, iodine should be a sheet anchor of the treatment. Those in which this condition has come on after an infectious disease require cessation from their work and a sojourn in the country, and to a place in which the various medicated waters, baths, tonics, etc. could be had under supervision. I have seen most admirable results come from the long continued use of radium emanation water, not giving more than from 5 to 10 thousand mache units a day. According to the eliminating ability of the kidneys and the skin, the use of water is in order. According to Hamburger,[6] much benefit comes from the intravenous injection of moderate amounts of theobromine sodium salicylate (Merck) and diuretin (Knoll). These are dissolved in distilled water so that not more than 2 cubic centimeters of total quantity of liquid is injected at any time. In my opinion this is not a necessary procedure because the attacks of pain can be controlled by the use of warm applications, a leucodescent lamp or a basket containing a number of incandescent bulbs over the abdomen. Small doses of morphine may be required during the attack and the drug that controls the pain in the head best is a combination of strophanthus and theobromine.

In the majority of these cases a severe routine which will not permit the patient to continue at his work is required. It is not uncommon when I observe distinct sclerotic changes to advise the individual to discontinue work or to change its character. Care is required by these people that they do not engage in what might be termed avocations which require labor of the brain rather than of the muscles. Thus, more physical exercise in the open, such as golf or bicycle riding, are most helpful.

Most important in this connection is it that one find the cause, remove that cause, or treat it when possible. In this connection, prominent in clinical medicine as a cause for this condition, is chronic excessive intestinal toxemia of the putrefactive and mixed forms. When present these require active treatment, dietetic, hygienic and perhaps bacterial. Diagnosis and treatment of these conditions have already been mentioned. Where no condition can be ascribed as the cause, the following diet may serve to good purpose in a general way. For an intestinal condition a diet according to the intestinal condition present is in order. Care must be observed that the individuals do not over-indulge in food. Small quantities and frequently repeated are decidedly safer than three meals a day. A general diet that may be used for arteriosclerosis is the following:

Breakfast, 7 A.M.—Fruit—The juice of an orange, grapefruit, stewed peaches or apricots, plums, pears, berries (raw or cooked), grapes, apples (baked or stewed).

Eggs—Soft boiled, dropped on toast or shirred, two or three times a week.

Broiled honeycomb tripe. Baked potatoes. Dry toast. One glass of milk or buttermilk, or a cup of weak coffee.

Dinner, 1 P.M.—Raw oysters. Thin soup (no meat stock). Broth of oyster stew.

Vegetables—Irish potatoes, lettuce, spinach, celery, tomatoes, string beans, squash, cauliflower, onions, asparagus, beets, carrots, cucumbers (cut thin as tissue paper). Green corn, green peas.

Dessert—Fruit, molasses gingerbread, crackers and cheese (Camembert, Brie, Roquefort). One cup of weak tea.

Supper, 6 P.M.—Eggs may be taken when not eaten for breakfast. Baked potatoes. Two or three times a week a luncheon may be made of a bowl of milk with crackers and baked sweet apples, or a purée of vegetable soup with bread and butter and fruit may be taken. One glass of milk or buttermilk. If constipated, some popcorn or bees' honey may be taken.

Suggestions.—Avoid skins and seeds. There is no objection to weak coffee or tea once a day. One glass of hot water should be drunk on rising, and cold at 11 A.M. and 4 P.M. At least five glasses of liquid should be taken daily. Do not drink during the meal. If losing weight you should have egg and milk lunches. Take at least two vegetables beside the potato with dinner. As a rule the fruit will agree best after the meal. Do not eat bread with dinner. Eat slowly and thoroughly masticate the foods.

References.

1 CABOT: Autopsy 3474. Case Records, Massachusetts General Hospital.
2 BROOKS, HARLOW: Am. Jour. Med. Sci., May, 1906.
3 SCHNITZLER: Wien. klin. Wchnschr., 11 and 12, 1901.
4 MARKWALD: Zeitschr. fuer prakt. Aerzte, vol. 9, 1900.
5 PAL: Die Gefæsskrisen, Leipzig, 1905.
6 HAMBURGER: "Arteriosclerotic Changes in the Abdominal Vessels."

CHAPTER XIII.

Anatomical Conditions.

DILATATION OF THE DUODENUM.

Of late considerable has been written on dilatation of the duodenum. Articles have appeared tending to draw attention to this condition and divers deductions have been made on the clinical aspect of the subject. Dilatation of the duodenum in all probability is not a distinct condition, and one that is acquired. Usually it is secondary or a part of another condition, for it must be remembered that the first part of the duodenum is, anatomically and physiologically, essentially a portion of the stomach. Thus it must be plain that conditions that can cause a dilatation of the stomach can likewise cause a dilatation of the first part of the duodenum as a part of the process in the pyloric region in which perhaps the pyloric sphincter could also be relaxed. As met with in clinical work, dilatation of the duodenum may be classified as follows:

1. Accompanying general gastric myasthenia or atony in which there may also be a relaxation of pyloric muscle; most commonly seen in cases of late atrophic gastritis.

2. Dilatation of the duodenum which is a part of an atony which comes from a long-standing gastroptosis of marked degree.

3. Those due to obstructive reasons such as adhesions springing from the gall-bladder and involving the second part of the duodenum, obstruction at the duodeno-jejunal angle, either because of bands, sharp angulation, or following gastroenterostomy.

Dilatation of the duodenum which is a part of marked gastric myasthenia, or gastric atony, is an uncommon clinical finding. It is true that in the average case of gastric atony more or less dilatation of the first part of the duodenum takes place, but this is never marked in degree. Usually the transverse measurement of the first part of the duodenum quite closely corresponds with the transverse measurement of the pyloric end of the stomach, and it generally can be noted that the pyloric muscle is intact and functionating. In X-ray plates of some of these cases, particularly those made in the upright position, it can be noted that the barium assumes a dependent position in the

duodenum, giving the appearance of relaxation. However, no obstruction or delay is ever met with, and if such were to be found it would be entirely in the stomach and would be minor as a duodenal condition, and not important as a clinical factor in itself so far as the duodenum is concerned. Most of these cases have chronic intestinal toxemia, or a neurological condition.

In these instances, other than some attention to the diet in special ways, the treatment would be for the mentioned conditions. In cases of atrophic gastritis the treatment for the dilatation of the duodenum and the relaxation of the pyloric muscle is that for the atrophic gastritis which will be found in detail in the volume, "Diseases of the Stomach." It must be said, however, that in cases of atrophic gastritis when dilatation of the duodenum is visible we are dealing with quite a late condition, and the results from any kind of therapy are ameliorative and not curative, although the patients do very well on proper diet and medication. After continued lengths of treatment, while it is never possible to have a return of normal gastric secretion, one might accomplish a reduction in the quantity of mucus which is secreted by the stomach, but the degree of dilatation in the stomach as well as in the duodenum is generally a permanent factor. Not uncommonly, however, one may see the return of the functionating power of a relaxed pyloric muscle. It is never active as in a normal stomach, but is distinctly more so than is present in the early instance of the case.

Dilatation of the stomach, which is a part of an atony which comes from a long-standing gastroptosis, can be seen in a small number of these cases and generally is met with where the stomach has become considerably dilated, not so much in its transverse dimension but in its long axis—namely a dilatation of attenuation. I have observed duodenums in these cases to be dilated to the extent of the transverse measurement of the pyloric end of the stomach some ten centimeters distal to the pyloric muscle. The condition in the duodenum is a part of the condition of the stomach and it does not require treatment of itself. Not uncommonly one finds also in these cases a dilatation of the right side of the colon, sometimes the transverse, and I have seen the colon to be dilated as far down as the pelvic margin on the left side. In a gastroptotic case with a markedly dilated duodenum it is always positive that one is dealing with a late condition and almost always one wherein a chronic intestinal toxemia is present. These people represent the emaciated, semi-invalided class who require long extensive treatment to receive any benefit. What is interesting in this connection is that, unlike atrophic gastritis

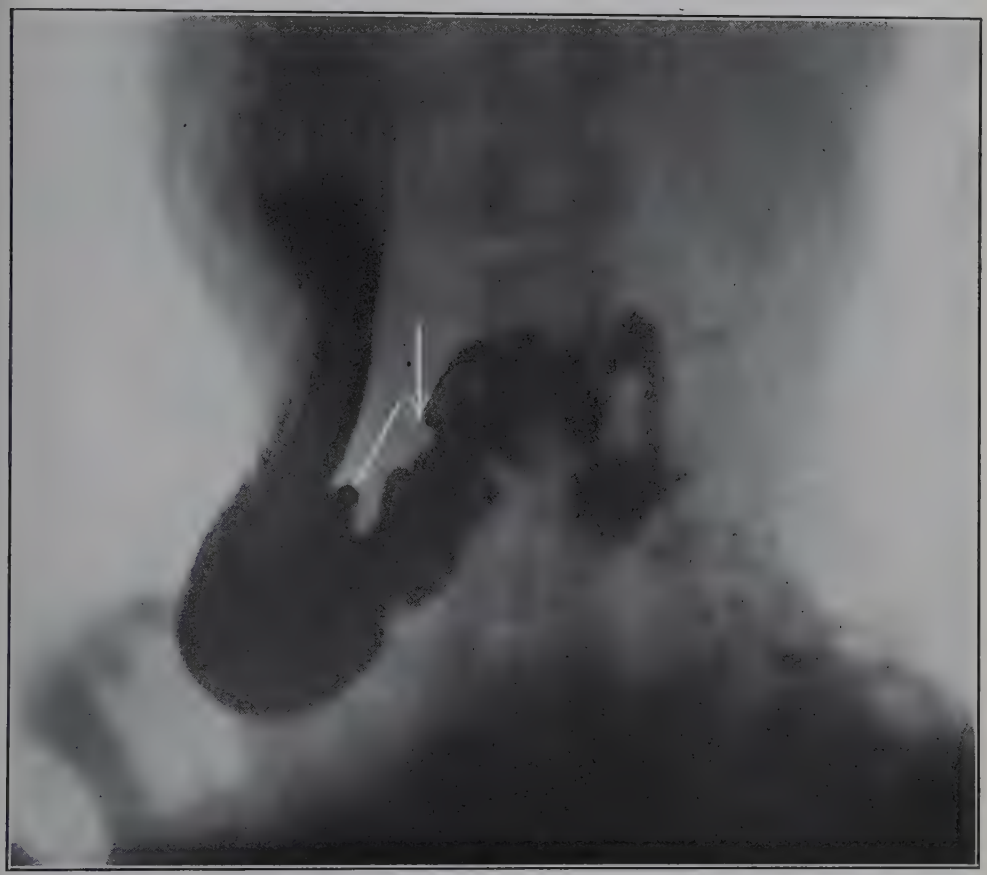
wherein the defunctionating pyloric muscle may be met with, the sphincteric action of the pyloric muscle is always preserved. Sometimes it is a little lax, the pyloric action not being as distinct, not as definite, or not as strong as in those stomachs of lesser degrees of gastroptosis, but at least it is clinically efficient. One must keep in mind that when a marked dilatation of the stomach is met with in gastroptosis and this dilatation has extended out into the first part of the duodenum, one is dealing with a case of gastroptosis closely bordering on the necessity for surgery for relief. However, with careful handling not a few of them are benefited by medical treatment, proper diet, support of the abdomen, rest tonic, tincture of time and the essence of patience.

Those instances of dilatation of the duodenum due to obstructive reasons, such as adhesions springing from the gall-bladder, usually present more or less dilatation of the stomach as well. While at operation in many of these it is proved conclusively that the duodenum is not distinctively obstructed, adhesions can, by anchoring the duodenum beyond a fixed point and interfering with its motility which is surprisingly rapid, cause more or less of a backing-up to occur. The same may be said of obstruction at the duodeno-jejunal angle because of bands, sharp angulations, or adhesions following gastroenterostomy, or, as is not uncommonly found, the stoma of the gastroenterostomy falling to one side and thereby becoming obstructive in the way of exit from the stomach into the jejunum.

When the adhesions are situated in the second part of the duodenum and have caused a proximal dilatation, it is not uncommon to see by way of test-meal examination that the exodus power of the stomach is approximately normal. It may be delayed an hour or so but never as distinctly as in definite pyloric obstruction. It is as if the interference with the peristaltic power of the duodenum, while the caliber of the tube is not definitely obstructed itself, can cause anatomical changes of moderate degree, these giving rise to definite symptoms of digestive distress. I have seen a number of those instances, due sometimes to reduplication folds of the peritoneum, non-inflammatory in type, in patients who give a minor history of duodenal ulcer. Most all of these patients gave the history of distress and sometimes pain some two or three hours after meals, relieved by the taking of foods or alkalies or any of the means which are employed to shut up the pyloric muscle. They therefore present a clinical factor in gastroenterological work and one should be on the look-out for them as they require operation for their cure, the results from operation being most beneficial and from medical treatment *nil*.

PLATE XXXVII

Dilatation of first part of duodenum due to general gastric atony and gastroptosis. (X-ray by author.)



Gastroptosis, dilatation first part of the duodenum. Penetrating ulcer in lesser curvature (arrow to right). Another nearer pylorus (arrow to left). Dilatation of duodenum due to gastroptosis. (X-ray by author.)

PLATE XXXIX

Showing the almost complete circle of the duodenum, exposed because the pyloric end of the stomach had to be pulled to the left in gastroptosis. In this case there were adhesions springing from the gall-bladder (arrow pointing) anchoring the duodenum, the first and second of which is moderately dilated. (X-ray by author.)

Adhesions anchoring the duodenum to under surface of liver (between arrows) and kinking at the duodeno-jejunal angle (obstructive, firm adhesions) causing dilatation in the third part of the duodenum. (X-ray by author.)

Idiopathic dilatation of right colon, about one-third of right transverse colon being involved. Case, one of long-standing intestinal toxemia. Result from medical treatment perfect. (X-ray by author.)

PLATE XLII

Case of idiopathic dilatation of the cecum and proximal half of the transverse colon, caused by pathology due to chronic intestinal toxemia. Complete recovery on medical treatments. Dilatation also of sigmoid. (X-ray by author.)

Moderate degree of megacecum due to long-standing intestinal toxemia. Recovery on medical treatments. (X-ray by author.)

PLATE XLIV

Megacecum, idiopathic, due to local pathology from long-standing intestinal toxemia. Complete recovery on medical treatments. (X-ray by author.)

When the obstruction for whatever reason is in the third part of the duodenum or the angle between the duodenum and the jejunum, usually the entire duodenum as well as the stomach becomes dilated. The case clinically then is one of a pyloric obstruction presenting all the cardinal symptoms of a largely dilated stomach secondary to the atresia. Such duodenums will be found very large in caliber and not uncommonly there will be a sacculation dilatation just proximal to the point of obstruction. These stomachs are usually stagnant, retaining bismuth or barium over six hours, sometimes as long as twenty-four, globular in shape, the gastric content being highly acid from concentration, and retention of food remnants is common. Generally large quantities of fluid are extracted from these stomachs which often is fetid from the presence of sulphureted hydrogen gas, and of course presents no lactic acid, Boas-Oppler bacilli or blood, the characteristic findings of pyloric obstruction of late cancer.

They are always operative cases, practically nothing being available for them in a curative sense without it. I have seen but one case of definite dynamic obstruction of the third part of the duodenum due to bands, kinks or angulations. Most all of the cases I have met with have followed a gastroenterostomy or some operative procedure in the upper abdomen. After a duodeno-jejunostomy these cases make a most perfect recovery. Some of the best results that I have seen have been in these instances.

IDIOPATHIC DILATATION OF THE COLON.

Idiopathic dilatation in the sense of the description of idiopathic dilatation that one finds in literature is a rare condition. There are various types of dilatation of the colon, non-obstructive in type, met with in the right side, or, more or less involving the transverse colon and undoubtedly acquired in type. They will be described, however, under the headline Megacecum. It is almost impossible to perceive any local condition in the colon or condition in the abdomen or general body which in an acquired way could bring on a marked idiopathic dilatation of the colon. Even in the most severe obstruction of the lower end of the colon where more or less dilatation exists the gut never becomes as large as one might assume it to be in a case of idiopathic dilatation of the colon.

On the other hand, it is not uncommon to see more or less dilatation of the colon, even of the entire colon, almost to the distal one-third, in cases of long-standing intestinal toxemia in which there is more or less atonic colitis. Usually, however, this is mainly on the

right side and may involve all the transverse as well, and I have seen many instances where dilatation extended almost to the pelvic colon, there being no obstruction anywhere, the condition undoubtedly being due to a long-standing catarrhal disease which involved the sympathetic plexuses, bringing about more or less myasthenia and atony of the musculature surrounding the gut. This is met with in cases of indolic or mixed forms of intestinal toxemia where these have been standing for years.

MEGACECUM.

In about two instances of every thousand Roentgenographical examinations of the abdomen what may be termed idiopathic megacecum is encountered. It is not uncommon to find large cecums due to bands, kinks or some condition distal to the cecum suggesting that the dilatation was a secondary or resulting process. However, not uncommonly, distinctly large right side colons are met with in individuals who have no obstruction distal to it, and there is no doubt in my mind that dilatation of the cecum and ascending colon may occur in an idiopathic way in an adult because of local pathology in the gut, it not being subsequent to obstruction. The misfortune in this connection is that since Hirschsprung's classical description of congenital idiopathic dilatation of the colon in 1886 many cases of idiopathic megacecum have been confused with the condition he described, which is definitely congenital while megacecum is generally acquired. More than that, Hirschsprung's disease is most commonly met with in infancy and childhood, and in the pseudo-mega colon occurring in adult life which occurs in Hirschsprung's classification the condition involves the lower colon, or the entire organ, and less markedly in the beginning (cecum). The condition herewith presented involves only the cecum and ascending colon and not any other portion of the intestine either above or below.

In all the instances that it has been met with by me it occurred in people between the ages of 40 and 60 years of age, individuals who had been well up to adult life, the condition occurring some time about puberty and probably not having any connection with the sexual organs. In my belief it takes years for the condition to become well established, and that it is due to bacterial processes represented in intestinal toxemia of chronic type. Exactly what these toxins are I am unable to say, but in all instances they have been cases of intestinal toxemia which present more or less local pathology of the right side which has been proven in few instances by careful sectioning, noting a change in the mucous membrane, an absence of

all the glandular elements, a zone of round-cell infiltration, almost complete absence of sympathetic nerve structure, myasthenia and atony of the musculature which may involve the entire right colon, sometimes even across the hepatic flexure in the proximal half of the transverse.

The clinical picture of the condition is obscure in a way. Definite ones are moderate constipation in which the bowels move every second or third day, and even if they move every day, a relative constipation exists. Definite relief from all symptoms for a day or two occurs after an active purgative, then there is a return after that taking from four to seven days, a slight degree of extension or fullness in the right side of the abdomen, noted particularly when the abdomen is thin and the walls relaxed, and the percussion of a large cecum markedly tympanitic. Then there is the clinical picture of a patient who has an intestinal toxemia to a marked degree, and the laboratory findings in the examinations of the stool and urine generally confirm it. Other notings are post-meal gastric distress, loss of weight, nervous breakdown, history of sanitarium treatments for this, and the observation of a large cecum by Roentgenographic examination. The dilatation usually involves the entire section to the hepatic flexure and may extend over the hepatic flexure involving the proximal half of the transverse. These people are usually of a debilitated, nervous type, although not always so. Apparently it is most common in women who represented all of my cases.

Treatment.—At the present time considerable debate is taking place as to the proper mode of treatment of these conditions. Much has been written by Lynch, Draper and others on the surgical treatment of right-sided pathologies of the colon. Various types of operation have been advanced. The partial colectomy, namely, the removal of the right side; ceco-sigmoidostomy, namely, the draining of the cecum into the sigmoid, and various other procedures. After a close analysis of such cases as I have seen I have come definitely to the conclusion that these conditions are medical in type. Mortality from surgical procedure is high and the results in way of benefit do not warrant it. Here and there are single instances of beneficial results, but considering the high rate of mortality and the many who are not benefited by operative procedure, I have become totally medical in my aspect of the subject. Of course, one must be careful that an obstruction of the colon does not exist to account for the dilatation, and that the case is represented in chronic individuals with a long-standing intestinal toxemia, and the general resulting state of health therefrom. When sure that one is not dealing with any

form of mechanical obstruction, the best mode of treatment is to find out what type of intestinal toxemia exists, employ the bacterial and dietetic treatment already mentioned, which, with sufficient rest in bed, massage, abdominal support, attention to the nervous system, correction of the constipation by dietetic means, the use of some form of purgation every four or seven days insuring a thorough cleansing of the intestinal canal, the patients become free from symptoms for various lengths of time, and sometimes for years. Up to the present time I have had twenty-one such cases under observation—the longest nine years, the shortest four years standing. There have been cases since then but these are not included. In every one of these instances I am satisfied that the medical treatment was distinctly beneficial, rendering these patients symptom-free, the patients themselves being well satisfied with the results which were accomplished. One must remember with these individuals, as with all chronic intestinal toxemia cases, that they are prone to recur and require attention from time to time.

HIRSCHSPRUNG'S DISEASE.
(Congenital Dilatation of the Colon.)

Instances of this condition have been known for over half a century, but attention was definitely drawn to it by Hirschsprung in 1886. Since that time numerous articles have been written upon it. The condition has been designated as congenital idiopathic dilatation of the colon, megacolon, megacolon congenitum, and giant colon. It will be noted by the reader of this volume that some distinctions are made in this classification tending to what I believe to be more or less definite entity.

What may be termed as Hirschsprung's disease, congenital idiopathic dilatation of the colon, is a condition which in all probability has its origin in utero. It is for that reason that I believe the term should be used only in those cases of stenosis met with early in life. The word "idiopathic" should not be used in connection with the subject. Among the various hypotheses suggested to explain this condition the classification of Barrington-Ward[1] may be given.

A. Mechanical:

1. An abnormally long mesentery, permitting torsion of the sigmoid (Barth).
2. Increased length of the intestine, especially the sigmoid, which throws the bowel into loops, and causes kinking and obstruction (Marfan, Neter).

PLATE XLV

Congenital megacolon (Hirschsprung's disease). (*Case.*)

3. Atresia of the rectum and anus (Treves).
4. Valve formation (Perthes, Roser).
5. Distention of pelvic colon, with meconium at birth, and valve action (Wilkie).

B. Neuropathic:

1. Neuromuscular defect of a segment of the intestine (Hawkins).
2. Paralysis of a segment of intestine (Pennato).
3. A lesion of the sympathetic (Bing).
4. Reflex spasm of the sphincters due to anal fissures (Fenwick).

C. Inflammatory:

Colitis becoming chronic.

D. Congenital:

1. Anomaly of development—dilatation and hypertrophy congenital (Hirschsprung).
2. Dilatation congenital, hypertrophy secondary (Mya, Concetti).
3. Hypertrophy congenital, dilatation secondary (Fenwick, Genersich).

It must be claimed that the above is far too complicated. It would have been best so far as the congenital condition is concerned to leave out classification B. and C. Further, classification A is questionable. I think that No 1 of classification B as offered by Hirschsprung explains most of the cases.

The condition is more often met with in male than female children in a proportion of over three to one. In occasional instances when the case goes on to adult life it would be questionable in my mind that even when a large dilatation is met with the condition should not be looked upon as secondary to some pathology. Even in that sense it is not an idiopathic dilatation but secondary. Most all of the cases met with are in the first year of life, or positively in the second. Occasionally patients may be met with as old as twenty or twenty-five years who have had the disease dating from birth or soon afterward, these can be classed as congenital.

Symptoms and Prognosis.—Of the symptoms, obstinate constipation and a distended abdomen are undoubtedly the most prominent. Practically all of the cases show these symptoms within three months, and sometimes even within a few days after birth. Usually one's attention is drawn to the condition by a marked constipation and an abdominal distention which develops soon afterward. This disten-

tion, however, may be delayed for a while, the constipation being existent for some time. The distention when begun usually reaches most unusual proportions, the skin over the abdomen becoming tense and glossy, with perhaps a decided contrast with the dry, sallow appearance of the skin of the face. The abdominal wall accompanying the distention may become thin—in fact so thin that one can see the peristaltic waves of the intestine.

Sometimes there are long intervals between attacks and the patient may be made fairly comfortable by the use of purgatives and enemata. Fecal vomiting and tumor are occasionally noted as prominent symptoms, attacks of diarrhea frequently alternate with constipation affording some degree of relief. Among the general symptoms may be noted emaciation, "auto-intoxication" and sometimes tetany. It may be of interest to note that children suffering from this disease sometime show a low mentality due to congenital brain defects, or perhaps absorption of toxins from the over-distended intestine.

The prognosis is very unfavorable because these little tots do not stand surgery well, and the rate of mortality is as high as 27 per cent. On the other hand, the rate of mortality regarding the medical treatment as expressed by Löwenstein in 1907 shows that of 59 patients treated he had 34 per cent. recovery and 66 per cent. deaths. Duval gives a mortality of 74 per cent. with medical and 34 per cent. with surgical treatment. The mortality is much higher in those under 5 years of age, and in the cases on record operative treatment in 14 infants (Dubois) was followed by 100 per cent. mortality; in 28, between 2 and 5 the mortality was 40 per cent; in 46, between 5 and 15 it was 26 per cent; in 41, between 15 and 50 it was 27 per cent; and in 14 patients over 50 it was 85 per cent. Of course, these records of Dubois's include cases of the non-congenital type because one does not see in adult life as large proportion of congenital (Hirschsprung's) type as this.

The complications that often set in are peritonitis, intestinal obstruction, bronchitis, bronchial pneumonia, hydronephrosis, and liver affections making the situation still more serious.

Treatment.—This disease has been treated both medically and surgically. The various medical measures are those usually resorted to for the correction of chronic constipation—namely, regulation of diet, cathartics, enema, massage, electricity, and the rectal tube. All these offer a temporary relief and may be used as preliminary treatment until the patient is in condition to undergo an operation, for there is no doubt that the consensus of opinion favors surgical intervention. Since the rate of mortality in infants under one year

of age is so high in surgical procedure, it may be wise to temporize with the case until the months of infancy are over. Of course if nothing worth while is being accomplished one is forced to resort to operation at once, but I have seen several children in whom the diagnosis was made in the first few months of life where we have been able to get along fairly well until the child's second year. At this time, operation can be done with slightly increased benefit in the mortality. What must be considered also is that when these little ones are operated upon operation should be done quickly, because they do not seem to be able to stand surgical shock. The first thing required is a preliminary colostomy, with or without that an enteroanastomosis or a resection, the latter being the operation of choice, this requiring at the same time an end to end or end to side anastomosis.

INTESTINAL DIVERTICULA.
(Peri-diverticulitis, Pericolic Abscess.)

With Graser's original paper describing a case of stenosis of the sigmoid due to chronic hyperplastic inflammation clinical interest in the subject began. Since that time numerous writers have written upon it and today it is accepted that acquired diverticula are of two varieties—the true and the false. True diverticula are merely wide-mouthed pouches whose walls contain all of the layers of the normal bowel. They are the result of an abnormal tendency to haustrum formation, and seem to be of anatomical significance only. False diverticula, on the contrary, are herniaform protrusions of the mucous membrane through the muscularis, and are of increasing clinical and pathological moment. Acquired diverticula may occur anywhere throughout the entire intestinal tract. They have frequently been seen in the duodenum in the vicinity of Vater's papilla. They are common in the colon and they cease abruptly at the first portion of the rectum. Occasionally they have been observed in the appendix, where they may be the seat of a condition which cannot be differentiated from appendicitis. They vary in number from several hundred to an isolated protrusion. Generally they are multiple.

While diverticula in various parts of the intestinal canal—mainly the duodenum— are capable of producing symptoms, nearly all of the cases that come under observation have been due to conditions resulting from diverticula of the sigmoid. It is for this reason that our interest in diverticula is almost entirely confined to the sigmoid region. Clinical experience shows that while diverticula may be met with more or less scattered throughout the entire large intestine, it is

Fig. 85.—Diverticula of the small intestine, showing the sacs of the diverticula; small sacs and the openings into them not seen.

in the sigmoid region that trouble usually occurs when the cases come under clinical observation.

The origin of the acquired form of diverticula is still a mooted question. A predisposition to diverticulum formation undoubtedly exists, and according to Graser and Sudsuki; this is common. This is confirmed in the operating room experience of any large hospital in which peridiverticular abscesses are not uncommonly seen. It is probable that the vascular theory accepted by Fischer, Koch, Hansemann, Berner, and others, and which can be corroborated by microscopical study, offers the explanation for most of the cases. They occur in the fenestra through which the vessels penetrate the muscularis and form the loci minoris resistentie. The vessels are surrounded by loose connective tissue which contains a variable amount of fat. As the individual ages, the fat increases, the connective tissue becomes weaker, and the fenestra are increased in size by atrophy of the muscularis. Intermittent stasis also stretches and increases the diameter of the fenestra. During periods of cessation of stasis, the caliber of the vessels diminishes, and loose space in the "vessel holes" results. These factors increase the predisposition. Accumulation of feces and gas incident to constipation, bowel atony, and abnormal decomposition act to a degree as determining factors.

Bland-Sutton believes that the appendices epiploicæ form points of lessened resistance because these in some way predispose to diverticula. Franke believes that developmental anomalies

are at the basis of diverticulum formation. Beer and Telling consider weakening of the muscular coat in adult life as the chief cause of diverticula. Age, to some extent, seems to be a determining factor. According to Telling the average age is sixty years. Sex also seems to play a part. About twice the number of cases are met with in the male as in the female. Obesity has been emphasized as a causative factor by Klebs, Mayo, and others, but it seems to be only an indication of involution processes and thus only a concomitant of weakness in the muscularis or in the "vessel holes." The physiological rôle of the sigmoid must be of some moment in promoting diverticulum formation. The feces are retained longest in this segment and its walls are incessantly subjected to changes in pressure due to variations in the quantity and quality of its contents. When the physiological retention of feces becomes excessive and constipation arises, the strain upon the gut walls is abnormal and any tendency to atony is aggravated. Localized weakenings are subjected to a determinative factor. It is probable that diverticula occur as often as 5 per cent. in autopsies in individuals aged 50 years or over. These minute herniaform protrusions of the mucosa range all the way in size from being discoverable only with the microscope, to processes the size of peas or grapes. They are conical, saccular, spherical or teat-like processes. The ostia vary markedly in size. Sometimes the diameter of the opening equals that of the average diameter of the diverticulum. Often the openings are narrow, and sometimes one must search carefully to find the mouth. Many of them are distinctly flask-shaped, and their necks seem to be constricted by the penetrated musculature. The coats of the diverticulum vary. Mucosa, submucosa, and serosa are constant. The amount of muscularis varies in different diverticula, and in different parts of the same diverticulum. The walls are thinnest at the tips of the processes.

Most cases of diverticula give no symptoms during life. No cases of acquired diverticula in the very young have been reported. The vast majority of the cases met with are those which have been examined by X-ray and the presence of the diverticula have been stumbled upon rather as a matter of surprise.

The morbidity from false diverticula depends on the following factors: Thinning of the coats; ulcerative and perforative action of retained concretions and feces; presence of pathogenic micro-organisms, and defective drainage due to (*a*) lack of an effective muscular coat, (*b*) closure of the neck by edema of the mucosa secondary to colitis or stasis, and (c) strangulation of the opening by kinking, torsion, or muscular action. According to Telling, the secondary

pathologic processes in diverticula are summarized as follows: 1. Thinning of the diverticulum wall. 2. Perforating action of the retained concretion. 3. The presence of micro-organisms and their toxins. 4. Inflammatory reactions of varying type and degree. With this data one could forecast almost with certainty the various kinds of cases that might be expected to occur clinically, and the list provides examples of nearly all of them. The following is a summary of the secondary pathologic processes to which sigmoid diverticula are liable: 1. Infection of the general peritoneal cavity, from thinning of the sac walls, without perforation. 2. Acute or gangrenous inflammation—diverticulitis. 3. Chronic proliferative inflammation with thickening of the gut wall and stenosis of the bowel. 4. The formation of adhesions, especially adhesions to (*a*) the small intestine, and (*b*) the bladder. 5. Perforation of the diverticula, giving rise to (*a*) general peritonitis, (*b*) local abscess, (*c*) submucous fistulas of the gut wall, and (d) fistulous communication with other viscera, especially the bladder. 6. The lodgment of foreign bodies. 7. Chronic mesenteritis of the sigmoid loops. 8. Local chronic peritonitis. 9. Metastatic suppuration. 10. The development of carcinoma. 11. Perforation into a hernial sac.

According to Mayo,[2] cases of diverticulitis are classified into four groups, as follows:

Group 1. Self-limiting Diverticulitis and Peridiverticulitis.—This group includes fleshy, middle-aged persons who present themselves with an acute sensitive tumefaction in the left iliac fossa. The mass gradually disappears in the course of some days, with restoration to health. The disturbance is due to irritation of fecal concretions, dead epithelium and other contents, in the thin-walled, narrow-necked sacs, which cause obstruction from edema and infection and penetration of bacteria to the peritoneal surface. There is marked tendency to relapse quite similar in character to that of relapsing appendicitis, and in the early histories of the patients of Groups 2, 3 and 4, it will often be found that several such attacks had occurred before the severe attack which necessitated surgical interference. That diverticulitis does not always produce trouble is shown by the relative frequency with which this condition is found *post mortem*, by the frequency with which diverticula of the sigmoid are a chance finding in the course of abdominal operations for other purposes, and by the frequency with which routine Roentgen-ray examination of the colon shows symptomless diverticula. One should not assume, therefore, that the presence of these diverticula, or even a single mild attack of diverticulitis which quickly subsides without obstruction or other

serious symptoms, necessitates operation. Patients of this kind are often poor surgical risks from other causes, such as obesity, and a considerable mortality attends resection, the only operation that really cures the disease. It is only in cases, therefore, in which the symptoms are serious or the disease becomes chronic or relapsing that operation is to be considered.

Group 2. Diverticulitis and Peridiverticulitis with Formation of Abscess Resulting in Enterovesical, Enterocutaneous and Other Fistulas.—This group includes those cases in which infections—either a developing peritonitis with abscess formation or the results of infectious processes which connect the diseased colon with the cutaneous surface, the bladder, or neighboring intestine—lead to the necessity for surgical interference. The rule is that if an abscess forms it should be opened and drained, but a serious attempt should not be made at the primary operation to remove either the infected diverticula or the section of colon which contains them. None of Mayo's patients died from a general septic peritonitis as the primary result of diverticulitis although such cases have been reported. The management of cases of complicated fistulas in which there are openings into the bladder and colon and to the cutaneous surface, and especially that most common type in which an internal fistula connects the bladder and the sigmoid, is very difficult. The obesity of the patient and the enormous amount of scar tissue which surrounds the fistulous tracts add greatly to the operative difficulties. In enterovesical fistulas the peritoneal cavity was opened, the fistulous tracts dissected out, and the openings in the bladder and colon closed with chromic catgut sutures. Rarely was the result immediately satisfactory. As a rule, a temporary fecal fistula to the surface formed after a few days, but when following operation, the bladder and sigmoid were kept separated by rolls of rubber tissue, and especially when the sutured opening in the sigmoid was protected by omentum, these secondary fistulas eventually closed spontaneously. A retention catheter was placed in the bladder for a week and a rubber tube fastened into the rectum following operation, to relieve tension.

Group 3. Obstruction.—In acute diverticulitis the obstruction is the result of infection and edema. Chronic obstruction is due to hyperplasia, adhesions and angulation—the hyperplastic stenosing type. The condition is practically identical with those in Groups 1 and 2, but the addition of the obstruction in these cases is so serious a feature that it seems best to classify them independently. It was most surprising, however, when the entire mass was dissected out and the diseased bowel laid open, to find so little actual obstruction. In

this group a tumor is usually found, and in 31 per cent. malignant disease coexists. The authors know of no more difficult differentiation than to foretell whether a given obstructing tumor deep in the iliac fossa of an adipose patient is diverticulitis or carcinoma or both. On a number of occasions they had gone down on such tumors which had been explored and pronounced inoperable by surgeons of skill and experience, and on dissecting the tumor out, found that it was diverticulitis without carcinoma and that the enlargement of the lymphatic glands, which had been one of the determining factors in estimating inoperability at the previous exploration, was due to sepsis. As a rule, the obstruction from diverticulitis is not quite complete. In this respect it differs from that of carcinoma, in which condition complete obstruction may be the first symptom of the disease. Several times, with great difficulty, they had dissected out a considerable portion of the sigmoid and found that the infection involved only one or two diverticula. Therefore the offending diverticula might have been excised and the patient spared the risk of the more serious operation. In two cases it was possible to excise infected diverticula successfully and separate the obstructing adhesions. On a few occasions a tumor was explored because of their inability to differentiate diverticulitis from carcinomatous disease. After a diagnosis of diverticulitis was established, drainage was instituted for the relief of the local peritoneal infection, with recovery of the patient. Fortunately, the Roentgen ray now furnishes the differentiation in a high percentage of cases. In acute obstruction it may be necessary to make a colostomy close to the obstructing mass so that the colostomy and tumor may be excised together at a second operation; or perhaps a better plan is to do a complete ileostomy for temporary relief, then resect later, and finally, as a third stage, restore the ileum to the cecum.

Group 4. Carcinoma Developing on a Diverticulum.—This group is of great interest. Among the 42 cases of resection for diverticulitis, Dr. Mayo met with 13 in which carcinoma coexisted. The carcinoma had such definite relationship to the diverticulitis as to make it reasonable to assume that infection and irritation by hardened fecal masses in diverticula were the cause of chronic irritation and precancerous change. The only known fact of importance in the etiology of carcinoma is its relation to chronic irritation. The term "precancer" is used to denote certain cell changes taking place in the area of chronic irritation which, if found in connection with invasion of the tissues, would be typical of carcinoma.

Griffin[3] found that for every sigmoid resected for diverticulitis, 7 had been resected for carcinoma. Mayo's statistics show that in 300

resections for carcinoma, 180 were for sigmoid growths. In 42 cases of diverticulitis of the large intestine, the diverticula were in the sigmoid in 36. It has often been pointed out that carcinoma of the colon, especially of the sigmoid, may progress very slowly. Cases have been reported in which a colostomy was made for the relief of obstruction due to supposed carcinoma; the patients lived for a number of years and then died with carcinoma of the sigmoid, a fact which apparently proved that the condition had been carcinoma from the beginning and that the natural course of the disease had continued for eight or nine years. This inference is unwarranted. They operated in several cases of this type, and on resection of the growth found carcinoma developing on old diverticulitis. In tracing the early history it could be seen that the diverticulitis had been the cause of the obstruction for which the colostomy was done and that the carcinoma had developed at a much later date as the result of the chronic irritation. In one rather remarkable case, a supposed carcinoma of the sigmoid, associated with a carcinoma of the transverse colon, was found on exploration. The left half of the transverse colon, the splenic flexure, the descending colon, and part of the sigmoid was removed, uniting the remainder of the transverse colon to the lower sigmoid. When the specimen was unraveled, it proved to be diverticulosis of 6 inches of the sigmoid. The carcinoma had developed on one diverticulum which had become adherent to the transverse colon, and had spread into the peritoneal, muscular, and submucous coats of the transverse colon without penetrating the mucosa. There was no carcinoma in the sigmoid, the malignant process being confined strictly to the diverticulum and the transverse colon. At the present time it is usually possible to differentiate between diverticulitis and carcinoma by the Roentgen ray since in carcinoma a filling defect will be found in addition to the less reliable findings of blood, pus, and mucus in the stool. With the sigmoidoscope one can differentiate carcinoma associated with diverticulitis of the lower sigmoid and rectosigmoid, but may not be able to demonstrate diverticula.

Some of the patients with carcinoma associated with diverticula, gave a long history of having had, at various times, inflammatory attacks with development of tumefactions which disappeared. After the carcinoma developed, the symptoms became more or less continuous. In reviewing a series of early specimens of sigmoid resected for supposed carcinoma, Wilson[4] found that three which had been resected years ago, before all specimens were subjected as a routine to microscopic examinaton, showed diverticulitis and no carcinoma. The association of carcinoma with diverticulitis leads to the con-

clusion that when a tumor appearing to be diverticulitis but without acute symptoms is found in the sigmoid or colon, and especially if the tumefaction only partially subsides and then continues as a chronic mass causing symptoms more or less marked, carcinomatous change is to be suspected and resection should be done. If there is definite obstruction at the time, or if the disease is advanced, the two-stage operation of Mikulicz, Bruns and Paul, as described by C. H. Mayo, may be adopted. Frequently, instead of leaving the tumor attached to the wound, one can cut the protruding bowel and tumor off, leaving the two ends of the intestine closed by a clamp on each, suturing the intestinal stumps into the wound behind the clamps and thoroughly covering the parts with petrolatum. After from twenty-four to forty-eight hours, or as long as the patient was able to stand the gas pressure from the complete obstruction, the clamps were removed. The suggestion of Peck[5] to leave the ends of the intestine closed for some hours until adhesion takes place has been of great value on a number of occasions, and permits primary union of the operative wound.

Symptoms and Diagnosis.—A sufficient number of cases have now been collected in the operating-room and X-ray laboratory to show evidence that most people having diverticula suffer no harm from them. Diverticula of the small intestine appear to be almost without danger. In practically all the cases where symptoms have arisen the diverticula have been situated in the large intestine, usually in the region of the sigmoid. The reason of this is that diverticula of the small intestine, unlike those of the large intestine, do not harbor feces partly because of the more fluid nature of the fecal content and partly because the openings are less likely to be constricted. The probabilities are that unless the diverticula become infected no trouble ensues. Such infection could take place from a fecal concretion which has become locked up in the diverticula, finally causing irritation, ulceration and a spreading of the infection by the lymphatics into neighboring walls of the gut to the peritoneum, or perhaps the ulceration may perforate the walls of the diverticula causing a local abscess or a localized peritonitis, which may develop into a general peritonitis, or it may adhere to some hollow organ, more often the bladder, discharge into it and cause a fistula, or the abscess mass may absorb spontaneously. One should always suspect cases of gas and feces in the bladder as having an origin from diverticula.

The symptoms of acute diverticulitis are pain in the left side, with the formation of an inflammatory mass which can sometimes be felt through the left flank. In this way the symptoms resemble

a left-sided appendicitis, having all the symptoms of appendicitis on the left side of the abdomen. The onset is usually sudden, with severe pains in the left lower quadrant, this generally going through to the back. Vomiting with chills and fever often take place. The pain is constant with cramp-like exacerbations, generally not relieved by hot applications, or after movement of the bowels. Sometimes a diarrhea ushers in the onset of the case. Usually there is no blood in the stools, the pain persisting and becoming worse as the days go on.

The abdomen is generally tympanitic excepting in the left lower quadrant where after a few days just inside the crest of the ileum a mass, seemingly tender on palpation, generally adherent, and showing well-marked boundaries may be made out. In the instance of a woman a hard mass may be felt high up to the left of the cul-de-sac, not attached or connected with the uterus, and apparently free from the adnexa. This mass is usually firm and only slightly tender on vaginal examination. On rectal examination the same mass may be felt. What is interesting is that on bimanual palpation its tenderness usually is extremely faint.

There is generally a rather sharp rise in temperature with perhaps a reaction after two or three days and a gradual increase, showing rather a septic character. The pulse is usually elevated to a moderate degree, and leucocytosis is commonly present. I am quoting here the picture that is most often seen—namely, that of peridiverticular abscess. The misfortune in some of these cases is that the individual is so obese that one has difficulty in making out a definite resisting mass on the left side.

In lesser forms of inflammation of a mild subacute or chronic type the pains may be moderate in extent, sometimes associated with bladder symptoms, the patient making a recovery in a few days. However, occasionally these pass on to chronic inflammation causing thickening in the intestinal wall or mesentery which forms a palpable mass which may be mistaken for a carcinoma, or adhesions to other loops of the intestine resulting in signs of intestinal obstruction, due to these adhesions or to a narrowing of the caliber of the intestines involved.

Other cases again are those of an acute inflammation or perforation due to ulceration of the walls of the diverticula resulting in a general peritonitis or a localized abscess, the peritonitis starting on the left side and perhaps becoming general. The instance of the perforation of a localized abscess into the bladder or externally on the left side with the formation of a fecal fistula has already been men-

tioned. It is not uncommon in these cases that carcinoma eventually develops on the diverticula. In addition to the symptoms caused by the above-mentioned conditions, instances have been reported of perforation into a hernial sac, the formation of a loose body or fecal concretion free in the abdominal cavity and the development into a metastatic abscess in the liver. It must be remembered that in a very few of these cases a persistency of tumor formations may exist or perhaps stenosis of the bowels from cicatrization and contraction leading to chronic obstruction, the first or the second being a mimicry of carcinoma.

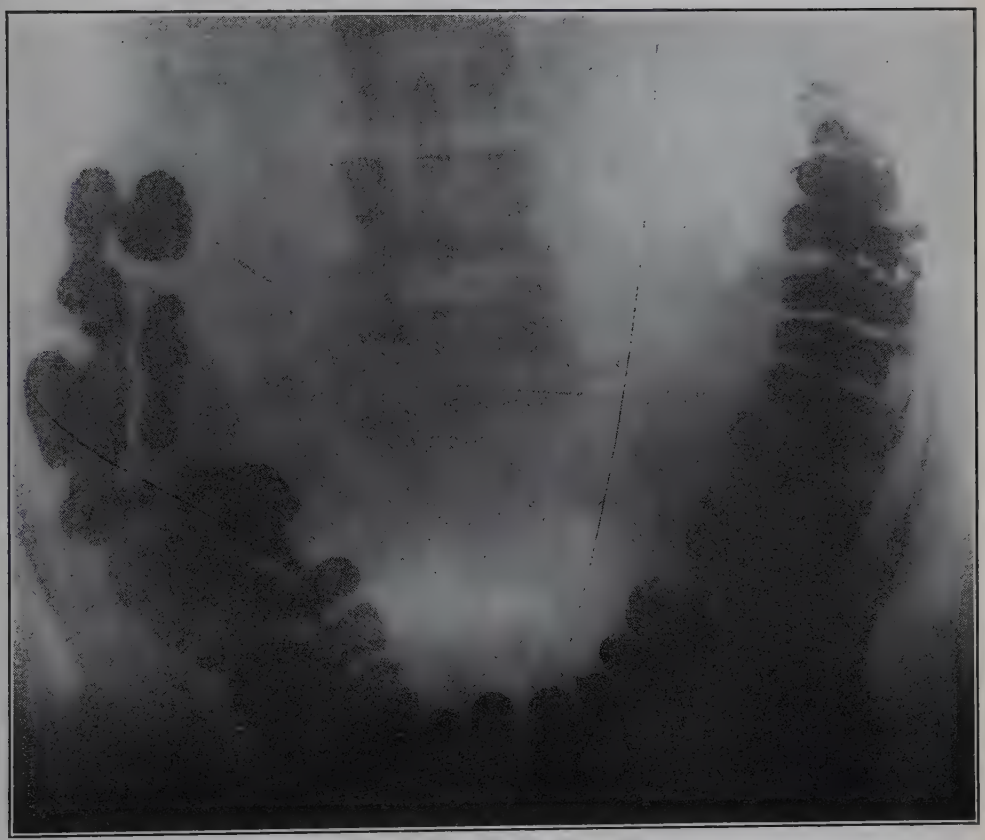
Finally, it may be said that in making the diagnosis of diverticulitis it should be remembered that, although the disease is more common than has been supposed, it is nevertheless comparatively rare and every left-sided pain is not always to be thought of as diverticulitis. As a matter of fact, the inflammations of the sigmoid are not yet thoroughly understood. Inflammatory tumors described by Braun and more recently by Myer which exhibit a general fusiform of local swelling about the gut somewhat resembling diverticulitis appear to have a mysterious origin, and one is at a loss to account for the chronic adhesions about the sigmoid occasionally seen during pelvic operations where origin from a salpingitis may be ruled out. In addition to the above one must keep in mind in the female the rupturing of a left-sided salpingitis or the occasional case of acute appendicitis with symptoms of pain referred to the left side. One is also to keep in mind the instances of chronic obstruction due to adhesion formation or other stenosing conditions about the sigmoid, as well as primary malignant disease and low grade infections of the gut and surrounding tissues.

Diverticula of the large intestine is most easily diagnosed by the X-ray, in which numerous small sacs containing the barium and the clysma may be held for days. Unfortunately this method of examination is most serviceable in instances of the symptomless types of diverticula. Generally these people do not come under observation for this distinct reason, they having been X-rayed for some other condition; because the diverticula themselves do not cause symptoms, until some acute condition has taken place. At the times of the acute symptoms the X-ray does not help us much, and one is rather loathe then to engage in it as the means of diagnosis.

Treatment.—Whenever, for whatever symptoms undertaken, the X-ray examination shows the presence of diverticula the patient should be apprised of their presence, because should trouble occur at any time it is worth much to the clinician to know that diverticula

PLATE XLVI

Coloptosis, flexures in place. (X-ray by author.)



Coloptosis, flexures in place. (X-ray by author.)

Mobile cecum, with colon in normal position. Some irregular peristalses in transverse colon. (X-ray by author.)

PLATE XLIX

Cecum mobile, showing also splanchnoptosis (coloptosis) with irregular, spasm, haustral contractions in the colon. (X-ray by author.)

Cecum mobile, showing considerably dilated cecum as well as it being freely movable by abdominal manipulation. Operation being required for the relief of the symptoms. (X-ray by author.)

Injected colon in a case of partial transposition. (*Case.*)

are present in the sigmoid. When faced with an acute condition one must judge the factors according to their severity. It is well to temporize in the beginning of a case unless one can be sure that a localized peritonitis and the possibility of a general peritonitis is imminent. In most instances, however, one goes on for a few days observing the individual, noting the temperature curves and the development of the septic factors. In the presence of an abscess it is always a fine point to decide as to when operation should be done. Of late my preference is to wait for several days because these abscesses are deeply situated in the pelvis in most of these subjects, and unless they are of considerable size one has some trouble in locating them. Such cases always are surgical, usually being followed by a fecal fistula after the incision of the abscess.

In the minor cases if the symptoms abate, one might withhold operation keeping the patient in an expectant sense a length of time. With numerous attacks of trouble in the left side resection would be in order. Just what are the indications for resection in hyperplastic sigmoiditis is a question difficult to answer, and depends upon the individual case. According to Mayo,[6] of 42 patients with diverticulitis, with and without carcinoma in whom resection had been done 14 per-cent. died as the result of the operation—that is, within four weeks. This rate of mortality he believes is due to a mistake in the early experience in believing that the tumefaction was due to carcinoma, and extensive dissections seemed to be indicated. Of late by paying more attention to differentiating between diverticulitis and carcinoma and not removing the mass when carcinoma could be ruled out better results were accomplished. In cases of obstruction Balfour[7] advises making a complete ileostomy close to the cecum after the manner of Brown,[8] bringing the end of the ileum out through the small opening on the right side and closing its distal end completely, thus entirely diverting the intestinal content from the large intestine. A cecostomy is made at the same time.

ENTEROPTOSIS.

The subject of enteroptosis has been presented fully in my work on the stomach under the term splanchnoptosis, which is by far the better term. Very little need be added to the subject here excepting to say that when the stomach is low in an abdomen and it has lost its ability of compensation, the transverse colon will always be found low with it. Since more or less descent of the liver takes place under these conditions, the right kidney and the hepatic flexure of the colon

usually descends, the splenic flexure of the colon almost always being found in place, although in a few instances it will be found low also. The condition is not uncommonly accompanied by chronic intestinal toxemia, which in my opinion can be the cause of the symptoms in these conditions. It is probable that factors which produce splanchnoptosis have nothing to do with intestinal toxemia, although intestinal toxemia can cause a loss of compensation in the digestive ability which is furthered by the low position of organs and their exhaustion by excess effort to empty themselves in these abnormally low positions. Therefore, it is necessary in a case of splanchnoptosis accompanied by a chronic intestinal toxemia to find whether the intestinal toxemia is primary and independent of the ptosis, or whether it is secondary to the ptosis. When it is secondary to the ptosis, treatment directed to splanchnoptosis is all that is necessary for the relief of such symptoms and general aspect of the case as come from the intestinal toxemia. But, on the other hand, if the intestinal toxemia is primary, treatment directed to the ptosis may bring about a degree of benefit, but the symptoms are liable to return in a relatively short space of time. For this reason a careful examination of the stools and urine are necessary to decide the presence of a primary toxemia which, when existent, requires treatment for its elimination after such treatments as have been conducted for the ptosis or during the time that the ptosis is having attention paid to it. There is no doubt that much of the opprobrium which has been cast upon the medical treatment of splanchnoptosis is due to the fact that a primary intestinal toxemia also existed and has not been cleared up or materially benefited, and also that many of the instances of surgery which have been beneficial in ptosis cases are due to the bringing about of a better mechanical state of affairs and in that way diminishing the toxemia. Even then, one not uncommonly sees cases in which surgery has been done, the stomach and perhaps the colon being in relatively much better position and many of the symptoms that the patient complained of continuing. There are instances of chronic intestinal toxemia which are biologic matters and for which surgery cannot offer much that is worth while.

CECUM MOBILE.

It is my opinion that cecum mobile are instances of chronic intestinal toxemia in which there is a dilatation at the very beginning of the ascending colon (cecum) in individuals who happen to have a long mesentery to the cecum. By it is understood that an ab-

normal motility of the cecum and lower portion of the ascending colon exists. This may be purely anatomical and give rise to no clinical symptoms, or as the result of the movement of the cecum kinks may be produced causing partial or temporary obstruction, ultimately giving rise to more or less atony and dilatation of the cecum with associated clinical symptoms.

The term was first used by Haussmann,[9] and since then the condition has been studied by Schultz,[10] Wilms,[11] Curschman,[12] and others. Since Wilms' article, cecum mobile has excited lively interest among the German and Russian internists and surgeons, and justifiably not so much among the French, English or American medical writers. According to Wilms, his attention was drawn to it because of so many cases of chronic appendicitis which had been operated upon and in which the symptoms failed to be relieved. In my opinion, as far as cecum mobile is concerned, it is a very poor premise offered as an explanation. Improper diagnosis would have been a better one.

Cecum mobile is not an entity, although it may be so pronounced as to produce certain local symptoms of its own. Usually it is one of the parts of splanchnoptosis in which the colon is prolapsed, which means that a marked degree or a section of splanchnoptosis is present. As was stated before, not all cases of splanchnoptosis are accompanied by cecum mobile for the reason that two things more are necessary, a long-standing intestinal toxemia plus a cecum with a long mesentery. To a considerable extent the subject is a congenital malformation of the meso-colon of the cecum, this being long enough to permit of the torsion and free mobility. Thus it is rather because of the long mesentery that a kink may form, usually occurring near or below the hepatic flexure, or there may be a reflection upward and forward of the cecum upon the ascending colon more like a volvulus of the cecum.

Symptoms.—There appears to be some difference of opinion regarding the symptomatology of these cases. According to Fischer the clinical picture is fairly uniform. Attacks of colic occur at irregular intervals, but with a general tendency to increase in frequency, severity and duration. Each usually begins with a longer or shorter period of constipation, and there is severe pain lasting for a few hours, about two according to Klose, or more rarely for several days, and terminating in a copious discharge of feces. During the attack there is loss of appetite and perhaps nausea and even vomiting; the temperature is either normal or, if fever occurs, it is slight. The leucocyte count is normal. A mass can be felt in the lower right quadrant about the size of a small apple, firm but not hard, and elastic but not doughy. Nothing can be felt on the left side. Tenderness is usually

present. Gurgling can usually be elicited. Posture has a pronounced effect upon the pain. If the patient stands or sits the pain is worse. It is relieved, and indeed the attack may sometimes be terminated, if the patient lies upon the back or particularly upon the right side. Predisposing factors are exertion and the indulgence of food that produces flatulence. During the interval the patient may be subjectively well, but usually symptoms of chronic colitis are present; that is, alternating diarrhea and constipation, mucus in the stools, and intestinal flatus.

According to my experience, cases of cecum mobile do not present any such clinical features as the above. There may be attacks such as have been described but these cannot be disassociated from other conditions which may occur in the colon, as in almost any case of chronic colitis without a cecum mobile. There can be no question, however, that more or less local pain or tenderness exists in these cases, and that they are prone to attacks of severe constipation, more so than is found in an average way. Not uncommonly there is a degree of distress described as being in the right flank or rather high in the abdomen on the right side. Usually the cecum can be palpated when a resistance would be noted. Not uncommonly there is a splash produced by transferring fluids up and down the cecum from the tips of the fingers of one hand to the other and back again. Tenderness on pressure exists in about one-third of all the cases.

The best method of examination is by the Roentgenographic method in which the filled cecum notably enlarged in its transverse diameter, somewhat low and free in the abdomen can be transported with a considerable range of motion on fluorescent screen examination. It is often found that the barium may remain in the cecum for two or three days (cecal stasis).

The differential diagnosis is as yet based rather upon theoretical than upon practical considerations. Cecum mobile is commonly mistaken for chronic appendicitis, and the distinction is rendered more difficult by the probability that appendicitis forms a frequent complication. There is a tenderness over McBurney's point, or what is more common, more or less general over the cecum. Constipation and local tenderness are frequent symptoms. The signs of distended or relaxed colon, the gurgling on palpation, and particularly the attacks of pain relieved by posture, when it can be obtained, may suggest abnormal mobility of the cecum or atony of the same. Mistakes have been made in diagnosing cholelithiasis, pseudo-appendicitis, various uterine or ovarian troubles for cecum mobile, and *vice versa*.

Treatment.—The majority of writers, Haussmann, Wilms, Klose, regard surgery as the only satisfactory remedy. In my opinion in the average case—in fact, in the great majority—it is about the last thing to do. What is required in these cases is to make a careful diagnosis taking all factors into consideration, particularly the one of the bacteriology of the intestinal canal. Treat the case about on the order of a case of splanchnoptosis, in which long rest and careful dieting, together with abdominal support will be in order and answers best after which, if a primary intestinal toxemia exists, this should be benefited or relieved according to the method of treatment already suggested. These individuals usually should be placed upon an anti-constipation diet such as the following:

The plan of the diet is to eat three meals a day with nothing between times. Food should be of the normal kinds and simply cooked.

Drink at least five glasses of water a day, preferably before meals and between them, not during the meal. Any of the customary foods can be eaten but do not take more than two eggs in a day and any of meat, fish and fowl but once a day, and then in very small quantity.

Milk, tea or sour wines are forbidden, and no food containing pits or seeds are allowed. Fruits, raw or cooked should be eaten morning and evening; the liberal use of honey and the use of milk sugar instead of cane sugar on your foods is advisable.

At breakfast each morning instead of a cereal, or with oatmeal, eat a handful of finely cut agar-agar with fresh cream. This can be purchased from the local druggist. During the course of the day eat one to three of the following gems, which should be baked twice a week:

Bran Gems: 2 cups of bran, 1 cup of flour, 1 cup of milk, ¾ cup of molasses, ½ teaspoonful of baking soda (dissolved in hot water), ½ teaspoonful of butter, ½ teaspoonful of lard, salt to taste. Bake in a slow oven forty-five minutes.

If it is not possible to take the bran gems, take one or two tablespoonfuls of wheat bran cooked in milk with cream and sugar.

At dinner, or before retiring, take a dish of stewed prunes cooked in milk sugar or honey. It may be found that apple sauce sweetened with milk sugar will work as well as the prunes. If so, you may alternate this with the prunes, eating one on one day and the other the next.

At certain times each morning and evening make an effort to stool. This should be persisted in even if no success is obtained in the beginning. Its object is to establish a regularity in time. However, at any other time when the desire for stool comes on respond to it at the earliest possible moment.

If a case of intestinal toxemia exists a diet may be called for according to the type of toxemia present. Usually more or less general treatment is required such as constructive tonics, perhaps the hypodermic use of cacodylate of strychnia and glycerophosphate of soda, and as is sometimes in order, a sojourn in the country and prolonged rest are advisable.

When the case gives a distinct colic history or there is a history of recurring attacks of appendicitis, operation is required. There are instances where after careful medical treatment and nearly all of them get well on this—that these symptoms continue and operation will then be in order. The operation first to be considered is the one advanced by Blake of cecumplication, Wilms' cecopexy being as good in its result. In my opinion the operation of ceco-sigmoidostomy for the purpose of draining these cecums, while beneficial in the average case and somewhat relieving the symptoms, is not a fair operation to perform. I say fair, because usually in a length of time, sometimes recorded as late as three years, the symptoms return and difficulty of obliteration of the anastomosis makes the original operation unfortunate. I believe distinctly that if operation is performed it should be an infolding plication or a cecopexy, not a make-shift operation for the drainage of the lower end of the cecum into the sigmoid. It is true that the first two require more surgical skill than the latter, but they meet the indications better and are fairer to the patient.

NON-ROTATION OF THE COLON.

(Left-sided Colon.)

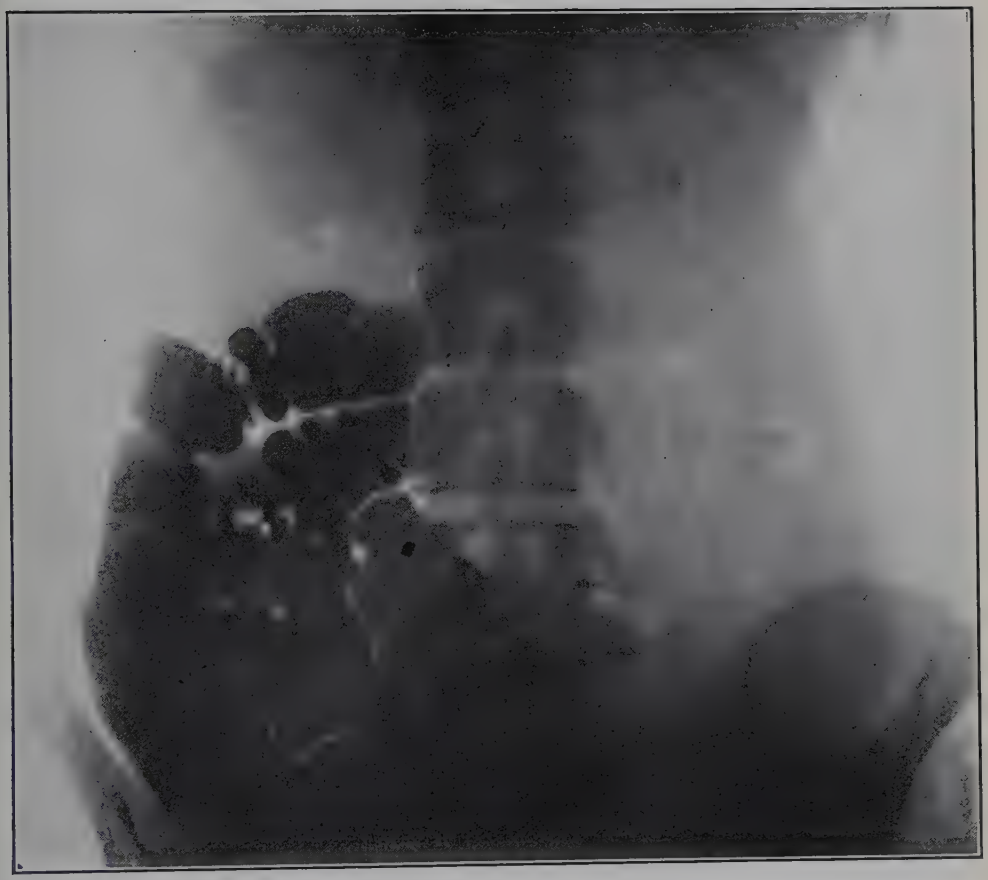
Failure of the colon to rotate, which usually happens about the seventh week of fetal life, is rather unusual. Very often non-rotation of the colon exists in patients who go through life without the condition being discovered until perhaps at autopsy.

Symptoms.—The instances where symptoms of non-rotation take place probably are due to the failure of the colon to empty itself and a limitation of its freedom of motion. Constipation is usually present and there is usually the history of more or less distention and colicky pains, together with indigestion, sick headaches, flatulence, nausea, and perhaps occasional vomiting. The people are generally neurasthenic in a chronic way, and always complaining whatever is done for them.

The condition is discoverable by X-ray examination, although, sometimes clinically it can be noted that there is a distention on the left side of the abdomen, or perhaps in the center due to the malplaced section of the gut. On examination it will be noted that the hepatic flexure is situated to the left anywhere from where its normal position would be to the opposite side of the abdomen. It may be that the descending colon only is in normal course, the entire left half of the abdomen containing the entire colon, flexures and all.

Treatment.—With these people it would be well to make an effort to meet such indications as exist symptomatically and have them wear

PLATE LII



Non-rotation of the colon. It will be noted that the entire colon is in the left half of the abdomen. The cecum and ascending colon is to the left, the transverse in the center, and the descending to the right. (X-ray by author.)

an abdominal belt such as is described in my volume on the stomach. If in such instances the symptoms are not relieved and a definite toxemia exists anastomoses may be in order. Anastomosis between the ileum and sigmoid should first be considered, and if this does not give relief more radical measures should be undertaken. Removal of the colon is comparatively simple in a non-rotation case, although the question of such a radical surgical procedure is a matter of the individual case and must be considered in the light of the symptoms and general condition of the patient. Two in which I had colectomy done got no benefit from operation. These people must be looked upon as congenitally deficient and poor subjects to promise much if any benefit from any procedures.

REDUNDANCY OF THE SIGMOID.

The sigmoid flexure, more properly called the pelvic colon by the French, is the segment which connects the descending colon with the rectum. It is of different lengths in different individuals, from as short as 15 centimeters (6 inches) to as long as 85 centimeters (32 inches). As a rule the longer the sigmoid the greater the liability of its being the seat of either functional or organic disturbance which often may be attributed to redundancy alone.

The full clinical significance of the sigmoid cannot be comprehended without due consideration of its relations. When redundant in the male, the sigmoid may lie in contact with the left ureter, the left iliac vessels and the left sacral nerves, the bladder, prostate and rectum, while in the female it might lie in contact not only with these structures (excluding of course the prostate) but also with the vessels in the left broad ligament, with the left Fallopian tube and ovary, and with the uterus itself. The degree to which these structures are influenced by superimposed weight of the redundant sigmoid is of course relative to the redundancy itself, to the incidental development of the meso-sigmoid and still more incidentally to the sigmoidal content.

In the normal person the sigmoid is chiefly a conduit for the less rapid transit of the intestinal content of the colon to the rectum. Usually after normal evacuation of the bowels it is relatively free from fecal content. The sigmoid is provided with an abundant absorbent apparatus and is connected with the larger chyliferous apparatus in the mesocolon.

Redundancy of the sigmoid occupies the second pathological group of sigmoidal conditions. The atonic catarrhal group occupying the first. It is very commonly met with in ptosis conditions wherein

the redundancy of the sigmoid is due to the same factors as cause an attenuating elongation of other parts of the hollow viscera of the abdomen. It is analogous on the left side with cecum mobile on the right.

Symptoms.—The symptoms of redundant sigmoid are variable. Probably the most constant one is constipation, with more or less colicky pains which are usually so indefinite that one's attention is not drawn to it. In the majority of instances I have observed that the colicky pains are in the very lowest part of the abdomen, usually transverse just across the symphisis pubis and not uncommonly referred into the back. These colicky pains are more liable to precede than attend effort at defecation, although they may be experienced at both times. The constipation is liable to alternate with diarrhea, especially in cases of so-called mucous colitis wherein there is a large proportion of mucus content in the stools. There is usually more or less pressure symptoms, partly due to pressure upon the sacral plexus, generally more marked on the left side although it may be present on both. Often these patients complain of bladder irritation, and sometimes in men there is a distinct distress referable to the prostate, while in women there may be a tenderness in the ovarian region, especially on the left side. Pain at the more fixed points of the colon, namely, the hepatic and splenic flexures, is often present because of the frequency with which the redundant and ptosed colon is a concomitant of the redundant and ptosed sigmoid. Pain just under the margins of the upper left ribs is especially significant of angulation and consequent retardation at the splenic flexure, a not infrequent finding in redundant sigmoid. This symptom is best elicited by percussion in the upper left lateral wall of the abdomen, the splenic flexure some distance posterior to the stomach being a point which should never be tympanitic in health; but is commonly so when conditions of retardation of the fecal current are present in the descending colon or sigmoid. A further point of physical examination is a dullness in the left lower quadrant and perhaps a large scybalous mass may easily be demonstrable on palpation.

Usually there is more or less tenderness on pressure over the sigmoid, demonstrating (both in the history and on physical examination) the presence of a low degree of sigmoiditis. When this sigmoiditis has existed for some time there not uncommonly is set up a low degree of inflammation which extends out onto the peritoneal coat and causes the development of adhesions, or the formation of bands which may be simple sources of irritation, or bind the sigmoid down at the brim of the pelvis causing a distinct obstruction at that point.

On careful sigmoidoscopic examination it may be possible to demonstrate the presence of a dry mucous membrane, paler than normal in color, and perhaps having more or less small particles of feces attached to it, and sometimes the attachment is very firm, the entire membrane having a glazed and dry appearance.

Occasionally there are various symptoms attributable to pressure upon surrounding organs. It may be that uterine displacement, or functional disorders of the ovaries characterized by pain especially at menstrual periods, is often brought about. More or less pressure upon the bladder is commonly met with in both sexes, caused probably from pressure within the abdomen and bringing about a stagnation in the inferior hemorrhoidal veins.

So far as my observation in redundant sigmoid cases is concerned I have never been able to definitely ascribe any general condition due to the redundant sigmoid. Such as are met with are those which could be easily attributable to any form of colonic disorder, being due simply to an intestinal toxemia, a ptosis, or other condition. It must be perfectly plain that if a redundant sigmoid is such a factor that it can interfere with the general state of health, the symptoms which would come would be essentially along the line of those met with in the toxemia, chronic constipation, and ptosis.

The conclusive method of examination is the Roentgen ray. By this means the barium-filled sigmoid, describing sometimes a most unusual course, although rarely presenting any definite resistance to the passage of fluid from the rectum upward, can be seen. One meets with so many cases of this disorder in gastro-intestinal practice that plates of the condition need not be included.

Treatment.—Speaking of the treatment of redundant sigmoid the attention of the reader should be drawn to the fact that in by far the majority no definite symptoms are due to the redundancy of the sigmoid itself. It is only in the minority of instances in which colicky pains due to the redundancy exist. In these, effort may be made and not uncommonly considerable benefit is brought about by simple means.

Where one is dealing with a condition like this the treatment for splanchnoptosis as outlined in my volume on Diseases of the Stomach would be in order. After the bed treatment, or during the time of an ambulatory conduction of a case, the patient should sleep with the lower end of the bed raised 12 inches or more from the floor. This modified Trendelenberg position has a tendency to benefit the redundancy of the sigmoid by taking the strain from its long mesentery, the patient being in this position at least part of each twenty-four

hours—namely during the sleeping time in bed. Sometimes it is wise during an attack of pain or colic during the day for the patient to adopt the Trendelenberg position or the knee-chest position for its relief. Usually a firm abdominal belt or a well-fitting corset which decreases the antero-posterior diameter of the abdomen below the transverse umbilical line is in order, this to be worn during the day.

The most important item of treatment is the dietetic correction of the constipation. For this one of the anti-constipation diets mentioned in this volume is useful. Continued effort should be made for the purpose of controlling the constipation by means of diet rather than laxatives by mouth or enemata. It has been my custom when laxatives are employed, as is usual when a distinct sigmoiditis is present, to use one of the saline cathartics for a while until the catarrhal condition of the gut has subsided. It will be remembered that the saline cathartics are resorbed from the stomach and upper intestinal canal and excreted by the lower part of the gut—namely the sigmoid and descending colon. In this way often much benefit is brought about in improving the local condition in the mucous membrane. In some of the cases small enemas are necessary and these are best given in the knee-chest position rather than the flat prone by means of the so-called colon tube (high enema). In other instances much benefit can be accomplished by the use of petroleum oil, particularly those rather high in paraffine, the best example of which is ordinary petroleum jelly (vaseline). This is taken by mouth before retiring, either heated to a fluid or taken cold in solid form.

Usually more or less constructive means to benefit the general state of health are in order. Of these may be mentioned the use of malt extract preparations, iron, strychnine, the phosphates, etc.

Of late considerable attention has been paid to operative procedure in these cases. Sigmoidopexy, ileosigmoidostomy, cecosigmoidostomy, and resection of the colon with even colectomy have been suggested. It is unnecessary for me to enter into a description of these operations, but I desire simply to say that in a very large number of instances of true redundant sigmoid accompanied by urgent symptoms due to the redundancy of the sigmoid itself the patients have gotten symptomatically well and are perfectly satisfied without having had any surgical procedure performed. However, there have been cases in which the local distress was so marked that sigmoidopexy was necessary, the resulting relief having been satisfactory. Some have claimed benefit from a resection of the sigmoid with lateral anastomosis and end to end, as well as other forms of procedures. I desire simply to state that the cases of redundant sigmoid having

definite symptoms due to the redundancy of the sigmoid alone which require operation are very few when the medical treatment has been well conducted. When obstipation exists operation is more commonly in order.

References.

[1] Barrington-Ward: Brit. Jour. Surg., 1914, 1, 345-360.

[2] Mayo, William J.: Jour. Am. Med. Assn., Sept. 1, 1917.

[3] Griffin, H. Z.: Diverticulitis of the Rectum, Ann. Surg., 1911, 53, 533-537; The Diagnosis of Diverticulitis of the Large Bowel, A Clinical Review of Twenty-seven Cases, The Jour. Am. Med. Assn., Sept. 14, 1912, pp. 864-866. Griffin, H. Z. and Wilson, L. B.: A Case of Carcinoma on Diverticulitis of the Sigmoid, Am. Jour. Med. Sci., 1909, 137, 661-666.

[4] Wilson, L. B.: "Diverticula of the Lower Bowel; Their Development and Relationship to Carcinoma," Ann. Surg., 1911, 53, 223-231.

[5] Peck, C. H.: Diverticulitis, Tr. New York Surg. Soc., 1910; Ann. Surg., 1910, 52, 264.

[6] Mayo, Wm. J.: "Diverticulitis of the Large Intestine," Jour. Am. Med. Assn., Sept. 8, 1917.

[7] Balfour, D. C.: Meckel's Diverticulum: A Report of Fifteen Cases, Jour. Minnesota Med. Assn., 1911, 31, 110-112.

[8] Brown, J. Y.: The Lane Operation; The Indications for and the Limitations of the Procedure, with a Discussion of the Principles Underlying it. Tr. South. Surg. and Gynec. Assn., 1911, 24, 137-150.

[9] Haussmann: Berlin. klin. Wchnschr., 1904, No. 44, p. 1153.

[10] Schultz: Case 14. "The beginning of the ascending colon lay deeper than normal." Case 19. "The ascending colon was so bent and doubled upon itself that the fundus of the cecum, directed upward, touched the portion of the transverse colon lying under the liver." Case 3. "The colon overlying the small intestines passed upward from the right hypochondrium to the umbilicus." Vesalius has pictured the reflection upward of the colon.

[11] Wilms: Deut. med. Wchnschr., 1908, p. 1756; Zentralbl. f. Chirurgie, Sept. 12, 1908, p. 247.

[12] Curschmann: Deutsch. Archiv. f. klin. Med., 1894, liii, 1.

CHAPTER XIV.

Neoplastic Conditions.

BENIGN TUMORS OF THE INTESTINE.

In the small intestine, tumors of any kind as a rule are pedunculated. The propulsive movements of the intestine influence the shape of tumors in its walls. Thus simple tumors such as lipomata, myomata, or sarcomata arising in the mucous membrane of the small intestine tend to become polypoid. The danger to be feared from the presence of such tumors is intussusception of the gut, for the tumor, acting as a foreign body, is urged along the bowel and may lead to fatal obstruction. The peristalsis of the small intestine also exercises molding effects on deposits of cancer, primary or secondary, within its walls.

Benign tumors of the intestines are very rare. There are quite a few instances of various forms of such tumors, in literature, but compared to the great mass of literature on carcinomata of the intestine, the contrast is striking. It is probable that more benign tumors of the intestine exist than are diagnosed, namely, tumors which are too small to cause obstruction, whereas on the other hand, the malignant tumors in their rapid growth soon cause symptoms which bring the case under medical attention.

Perhaps the most common of the small and large intestine tumors are the intestinal lipomata of the submucous variety. They need not grow very large in size, one weighing not more than 60 grams would be sufficient to completely obstruct the small intestine and bring on an acute attack of obstruction or those of chronic invagination.

Dewis[1] in 1906 reviewed the literature on intestinal lipomata and found but 44 cases reported. The situation of the tumor in these cases was as follows: duodenum 6, jejunum 4, ileum 7, cecum and colon 5, sigmoid 5, rectum 6, small intestine 2, intestines 1, unknown 6. In a case recently reported by Goodall[2] in which there was a long-standing history of many years of illness, he draws the following conclusion: "1. It is necessary that one be entirely familiar with the clinical story as often the important features are only brought out by direct questions. 2. That in all chronic abdominal diseases this

condition should be excluded before a hopeless prognosis is given, and the disease allowed to run its natural course."

Other tumors that may be found are fibrinous, or those of the fibromyxomatous type. Of these likewise but few are on record, the rule being that many of the cases are not diagnosed until intense obstruction or intestinal invagination of the chronic type has taken place.

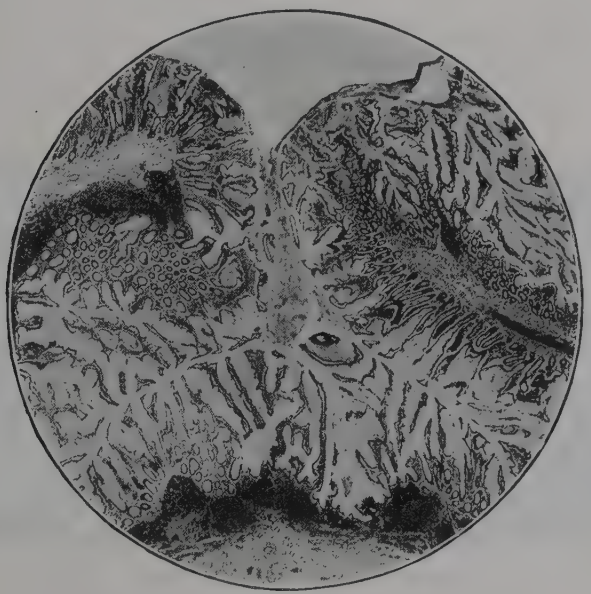


Fig. 86.—Adenoma of cecum. ×11.

INTESTINAL INVAGINATION.

Since generally tumors of the intestine are not diagnosible excepting when intestinal obstruction or chronic invagination has taken place, and intestinal obstruction will be found treated elsewhere in this volume, it may be well to only give a resumé of the symptomatology met with in chronic invagination cases.

As a rule it is found in middle-aged subjects, the history usually being that of a violent attack of diarrhea without known cause and which lasts for a few days. This often is followed by soreness which persists for a few days or a week and then a return to normal, when perhaps in a month or two a second attack of diarrhea comes on. With subsequent attacks of diarrhea a constipation ensues with which there is usually more or less frequent attacks of abdominal pain of a

crampy nature. At the time these pains are on there is usually a rumbling or gurgling sound, expressed by some patients as if something were pressing through the bowel. With this history, and examining patients during the attacks of pain, it may be noticed that the abdomen bulges, usually on the left side, a lump, perhaps quite hard, being palpable. At these attacks vomiting may be present and the case may look like one of intestinal obstruction when suddenly for no apparent reason loud squirting sounds are heard and the bowel becomes patent, a movement soon following.

When chronic invagination of the intestine has taken place a mass may be palpable or it may not. Careful X-ray examination may

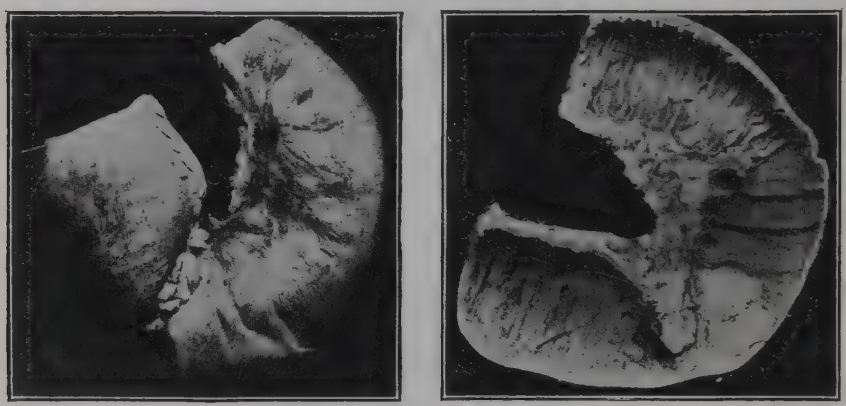


Fig. 87.—Chronic invagination of the ileum. (*Goodall.*)

reveal normal exodus time in the stomach and normal transit through the patent gut up to the point of the invagination. The head of the barium or bismuth may pass the invagination area, giving all the manifestation of a normal transit, but plates made at subsequent times will show that there is a point in the abdomen where the barium or bismuth seems to reside for as long as two or three days. However, in most of the instances definite obstruction in some one part of the gut will be noted.

INTESTINAL POLYPOSIS.

There is some close relationship between polypi and cancer of the intestine; such as exists between warts and cancer of the skin. Intestinal polypi, like cutaneous warts, sometimes appear spontaneously. Warts or polypi occasionally occur in vast numbers. Polypi may be limited to the rectum or extend throughout the colon. The

rectum is a common situation for them, and they usually occur in the interval between the fifteenth and thirtieth years. They give rise to bleeding, diarrhea, and pain—symptoms suggestive of cancer. Intestinal polypi may be flattened processes of mucous membrane, dotted with the orifices of follicles. Some are long and narrow, others clavate. There is a variety of polypus often solitary, fairly common in the rectum of children and young adults. It resembles a cauliflower, and may be pedunculated or attached to the mucous membrane by a broad base. The glandular elements in such polypi consist of columnar epithelium, and the whole cluster recalls a colony of anemones on a rock. When sessile polypi of this kind occur in adults, their removal is sometimes followed by recurrence and occasionally by cancer. In children glandular polypi are invariably benign. Some intestinal polypi are as rich in epithelium and as delicate as vesical papillomata. As a rule they are not large enough to cause intestinal obstruction or chronic invagination. It is my opinion that they start as a colitic process, the polypi being the result of ulceration. For more information concerning this condition refer to Colitis Polyposis (Virchow) found elsewhere in the volume. Although intestinal polyposis may be met with anywhere in the intestine it is most common in the sigmoid and rectum. Here it may last for many years, the symptoms being that of a colitis with attacks of diarrhea alternating with constipation. If the mass is low enough there may be troublesome tenesmus, and if high enough, intestinal obstruction may occur. A rather constant symptom is blood in the stool but a direct passage of fresh clots is rare. The blood is combined with a discharge of thin mucus which may be very abundant. The diagnosis is based upon direct examination by the proctoscope. This should be done in all instances of young people having the above history.

If there are many polypi they are widespread over the area of as much surface as can be seen by the proctoscope and medical treatment is of no avail. Essentially a diet, rectal irrigation, washing out with astringents are about all that can be done. Solutions of tannin serve a good purpose and those of nitrate of silver are also useful. Surgical treatment consists of shelling out all the polypi that can be reached. Where many polypoid growths are met with they are never limited to the rectum alone and the shelling procedure has led to fatal bleeding in some instances. However, if there is just one or two small-sized polypi that can be seen after examining the gut as far as the instrument will reach and these give symptoms, which they rarely do, it is wise that they be operated upon. The operation is that of extirpation which can be done through the endo-

scopic tube. In marked cases of polyposis, colectomy, partial or complete, is indicated.

CARCINOMATA OF THE INTESTINE.

Primary cancer of the small intestine is rare, while that of the large intestine is comparatively common.

THE DUODENUM.

Of the entire small intestine the duodenum, which is the shortest part, is most frequently affected. While the time-honored division of the duodenum into first, second and third parts may serve for anatomical purposes, it is inconvenient from a pathological standpoint. It is more proper, following Sherren, to call the portion above the bile papilla the supra-ampullary segment, the portion containing the bile papilla the ampullary segment, and the remainder the infra-ampullary segment.

As is well known, duodenal ulcer most commonly occurs in the supra-ampullary segment, namely in the first two centimeters of the duodenum, yet it is most rare to meet with carcinomata in this area. It therefore is most logical to believe that chronic duodenal ulcer is not a precancerous condition.

Cancer arising in the mucous membrane around the bile papilla is known as circumampullary cancer, in order to distinguish it from cancer arising in the ampulla. Both forms lead to jaundice and distention of the gall-bladder. There is no doubt that cancer of this section of the duodenum would often escape detection if it were not for the obstruction it offers to the flow of bile from the common duct. Cancer of the ampulla arises in the mucous membrane of the duodenum around the bile papilla which differentiates it from cancer arising above the ampulla which would block the common bile duct but not the pancreatic duct, whereas cancer arising in the ampulla blockades both ducts. Accordingly this mechanical interference with the outflow of bile and pancreatic juice can in the ampulla region present exceptional features. It is a rare disease, usually circumscribed and shows little tendency to invade adjacent structure or to disseminate. Nowhere in the body does so small a lesion lead to such grave interference with digestion. A cancerous growth sometimes no larger than a small cherry will block the outflow of bile and pancreatic juice causing intense jaundice and great emaciation. It leads to dilatation of the bile ducts, extra- and intra- hepatic, and enormous distention of the gall-bladder. In spite of its local nature it quickly destroys

life. When cancer of the ampulla gives rise to jaundice death usually follows in six months. Cancer of the ampulla, like cancer of the common bile duct, differs from this disease in the gall-bladder by being rarely associated with gall-stones. Very few instances of cancer of the ampulla have been recorded, the total number probably not being more than 200.

The common place for cancer of the duodenum is in the infra-ampullary section, and especially at the part where the duodenum is crossed by the superior mesenteric vessels. Cancer in this situation is usually of the constricting type. When the infra-ampullary duo-

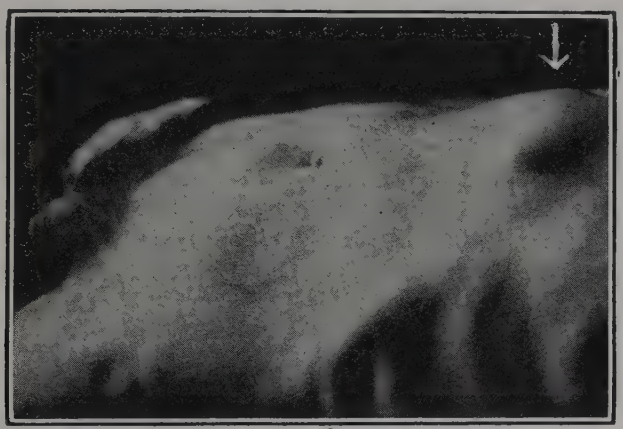


Fig. 88.—Visible peristaltic movement of the stomach occurring at the end of peristalsis of the small intestine in the case of carcinoma of the ileum. The onset of the movements began in the right iliac fossa in a small elevation. This travelled upward on the right side of the abdomen for a short distance, coursed transversely and slightly upward in the ileum, then disappeared in the jejunum deep and higher on the left side, the whole movement finally terminating in a peristalsis involving the entire stomach. Tracing the stomach movement, the slant from the left costal margin is seen with a peristaltic wave in the upper part of the body of the stomach just below it. Below this is a concave sulcus terminating in the peristaltic wave below, which wave can be traced around the umbilicus in the corresponding pyloric region. At the very height of the gastric movement a loud gurgling was audible from the fluid coursing through the stricture in the ileum, and a severe pain was present. Arrow points to the right costal margin.

denum is narrowed in this way it gives rise to signs similar to those seen in pyloric obstruction, but the vomited matter is extremely offensive, because it contains, in addition to the contents of the stomach, chyme that has been submitted to pancreatic digestion. The effects of the occlusion on the duodenum are also remarkable, for it becomes enormously dilated and like part of the stomach.

THE JEJUNUM AND ILEUM.

As was stated in connection with benign tumors of the small intestine, tumors of any kind in this section of the gastro-intestinal tract, as a rule, are pedunculated and commonly occasion intussusception. This is the rule in carcinoma of the ileum with the exception of the lowest part and here the carcinoma is liable to become fixed to the posterior abdominal wall, in that way preventing invagination

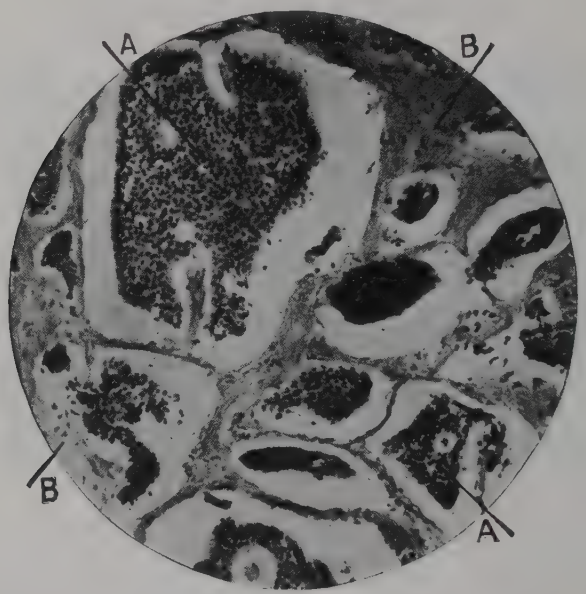


Fig. 89.—Photomicrograph of gland situated at the root of the mesentery, showing hyperplasia of the gland substance with carcinomatous infiltration at the hylum. Cortex showed a proliferation of lymph cells, and there is a general proliferation of lymphoid tissue throughout the gland. *A*, Masses of carcinoma cells. *B*, Hyperplasia of gland substance. ×200.

and presenting most definite intestinal obstruction. It is at the ileocecal junction that primary cancer of the ileum is most often met with, and as I stated above, is usually obstructive. However, in some ways primary cancer of the small intestine may resemble that in the colon because sometimes the cancer is of the annular constricting type or it may sprout like a cauliflower into the bowel. There is also a massive form which envelops the intestine with a large collar of new growth.

Fig. 90.—Secondary carcinomata of the mesenteric lymph glands.

CANCER OF THE COLON.

This is very common and it may arise in any part of the colon from the ileocecal valve to the anus. It is more frequent in some parts of the large bowel than in others; it being less frequent in the cecum than in the pelvic colon and rectum, and more common in my experience at the flexures than in the transverse or descending limb. Its usual form is columnar in type. Little is known of the early stages, because it gives rise to no definite symptoms until it mechanically interferes with the bowel or becomes septic. When well advanced it may project into the lumen of the bowel as a cauliflower-like mass. Its surface then is liable to become abraded and infected with pathogenic micro-organisms, especially the colon bacillus. This exuberant form is common in the cecum and ascending colon. In the transverse, descending, and especially the pelvic colon the atrophic, or constricting, form of cancer prevails.

Primary cancer of the vermiform appendix has been recorded by many observers. In some instances the tumor was so small that it would require a magnifying glass to see it. In a perfect case the carcinoma would be met with in the lumen of the appendix growing in an exuberant way usually blocking the caliber of this portion of the gut. I have seen instances of carcinomata sharply bunched about the appendix, involving the structure so that the appendix could no longer be found, the entire mass being plastered on the site of the cecum that the appendix occupied. In late cases carcinomatous masses above the appendix may extend to various locations in the abdomen.

Cancer of the cecum is not always easy of recognition, because infective conditions are common in this part of the intestine. Pericecal abscess is sometimes associated with cancer of the cecum. The matter is further complicated by infective conditions of the vermiform appendix and the occurrence of stercoral ulcers secondary to cancerous obstruction of the colon. Some instances of hyperplastic tuberculous disease of the cecum and colon in its naked-eye appearance are indistinguishable from cancer, and there are instances of actinomycotic infections and simple plastic inflammatory conditions of the cecum where the cecum has been excised on the supposition that it was carcinomatous.

Cancer may arise in any part of the transverse colon, or the transverse colon may be secondarily affected because of its close proximity to the gall-bladder and the stomach, from which cancerous growths may invade the colon, or *vice versa*. In some individuals,

PLATE LIII

Carcinoma of pelvic colon. (*Case.*)

PLATE LIV

Lymphosarcoma of the colon. (*Case.*)

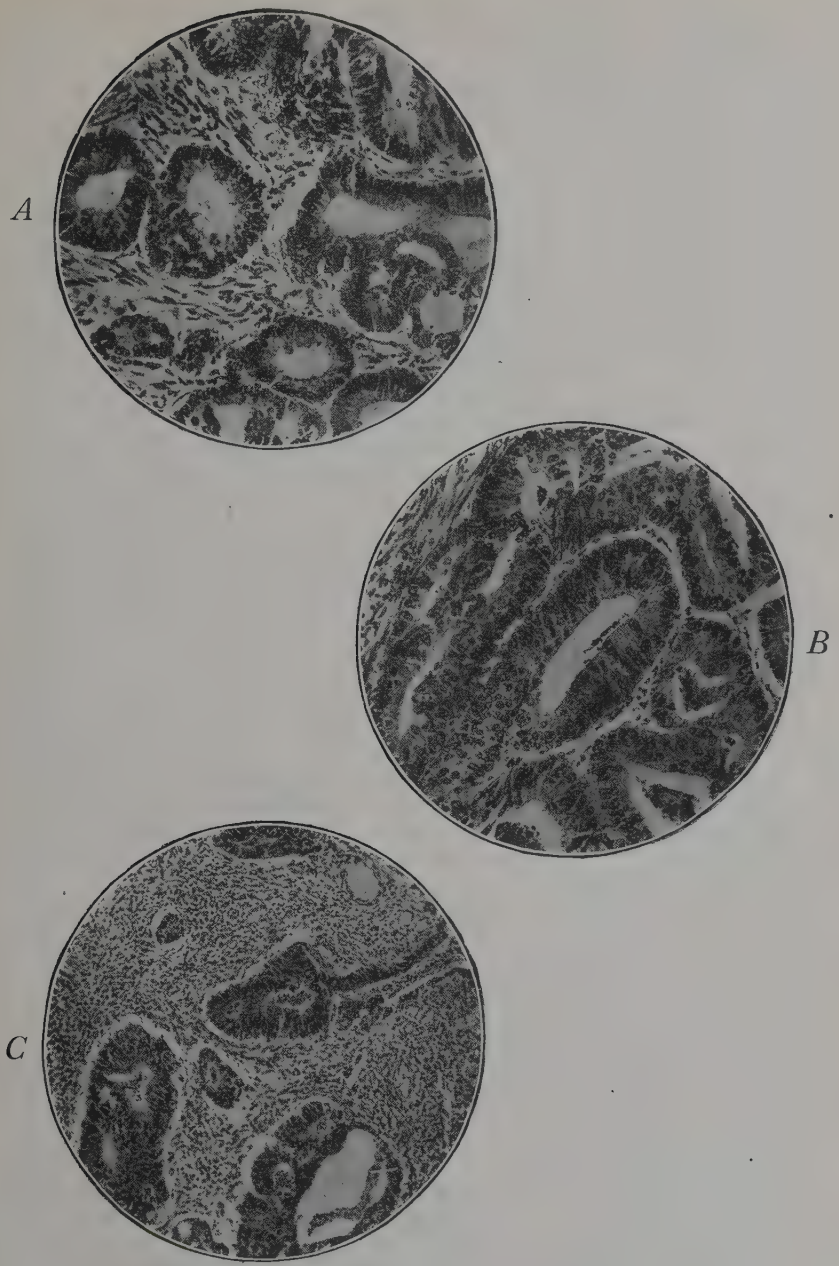


Fig. 91.—*A*, Rectal carcinoma, cylindrical epithelioma, involvement of muscularis. ×200. *B*, Rectal carcinoma, cylindrical epithelioma. ×200. *C*, Rectal carcinoma, cylindrical epithelioma. Sclerotic form and resembling inflammatory infiltration. ×112.

particularly in women, the transverse colon occupies a very low position in the abdomen, perhaps being as low as contact with the fundus of the uterus. Hence in carcinoma of the body of the uterus in which the transverse colon is involved it is impossible to say, whether the cancer arose in the colon and implicated the uterus, or *vice versa*. As a rule carcinomata of the transverse colon are discovered accidentally, generally at the time of operation for the relief of an acute or chronic cause of intestinal obstruction.

It has been the custom to call that portion of the colon between the splenic flexure and the limits of the false pelvis the descending colon, and the length between this point and the rectum the sigmoid flexure. In recent surgical writing the sigmoid flexure has been anatomically separated into two. The portion attached to the iliac fossa without a mesentery is known as the iliac colon. The remaining portion forms a loop completely surrounded by peritoneum and attached by a mesentery to the side wall of the pelvis. This is the pelvic colon. It terminates near the third piece of the sacrum, and at this part the peritoneal investment ceases to be complete. The occurrence of cancer in these three sections of the colon varies widely. It is very common in the pelvic colon, in the proportion of about 10 in the pelvic colon to 1 in the descending colon.

The mobility of the pelvic colon often leads to difficulty in diagnosis when it is occupied by cancer, for the weight of the growth causes this colic loop to sink until the cancerous portion rests on the floor of the pelvis. This brings it into close relation with the rectum and the bladder in men, and in women, with the uterus, ovaries and vagina. A cancerous growth in the colon is often mistaken for an enlarged ovary, a distended tube, or a subserous fibroid.

The length of the rectum is but one-twelfth the length of the colon from the cecum to its termination in the rectum. Yet cancer occurs more often in the rectum than in any other anatomical segment of the bowel. The rectum lies entirely within the pelvic cavity, its narrowest portion at the junction with the pelvic colon at the level of the junction of the second and third sacral vertebræ, and ends in the pelvic diaphragm. Its terminal portion is dilated, and called in consequence the ampulla.

Cancer of the rectum may start in any part of the mucous membrane of the rectum, but a common situation is just above the anal canal, and another favorite spot is at the junction of rectum and pelvic colon. The former situation is within reach of the examining finger; the latter is beyond its reach, but easily within reach of the proctoscope. Rectal cancer consists of glandular recesses lined with

tall columnar cells embedded in a stroma of dense connective tissue. In order to make out the nature of the growth, sections should be taken from the margins of the tumor, because the deeper parts are much altered by ulcerative and necrotic changes. In many cases of rectal cancer, judging merely from the appearance under the microscope, it would be difficult to determine whether a section was prepared from an adenoma or a carcinoma. But it must be borne in mind that the adenoma remains restricted to the mucous membrane, whereas in cancer we find the glands with their characteristic columnar cells interspersed among the muscular fasciculi of the gut-wall. The proportion of connective tissue varies greatly. In some cancers the glands are closely set; in others they are ill formed, arranged irregularly and embedded in an abundance of connective tissue. Occasionally collections of lymphoid tissue are observed. Because of bacterial invasion, cancer in the ampulla of the rectum may have raised edges, quite an area being plaque-like in shape. On the other hand, cancer of the pelvic colon is often of the constricting variety, and this variety is common in the section of the rectum immediately above the ampulla. Cancer is more frequent in the ampulla than in the anal canal, or at its junction with the pelvic colon. In all forms the disease infiltrates the muscular as well as the submucous tissues, and extends beyond the confines of the bowel into adjacent parts and involves peritoneum, pelvic connective tissue, prostate, vagina, bladder and sacrum. Ulceration occurs early. The disease is spread by lymphatics and by veins. The lymphatic vessels from the rectum accompany the hemorrhoidal veins, they conducting the growth to the lymph-nodes in the pelvis, then to those at the brim of the pelvis along the course of the iliac vessels, and onward to the set around the celiac axis. In the last stages of the disease very extensive infection of the lymph-nodes exists, and occasionally the thoracic duct is converted into a solid cord and enlarged lymph-nodes appear in the neck above the left clavicle. The dissemination of rectal cancer is effected mainly by the portal circulation, and the liver becomes the depository for the cancerous emboli. Sometimes cancer in the rectum produces very little disturbance, and is unsuspected until enlargement of the liver and perhaps jaundice lead the patient to seek advice. The nodular condition of the liver then prompts the surgeon to examine the rectum and the cancer is discovered. In other instances the cancer appears in the liver, lungs, kidneys and bones, which cancerous deposits display the Lieberkühn's glands with their tall columnar epithelium. It is because of this striking character of these columnar cells found in remote parts of the body that at

post mortem pathologists are now paying more attention to the presence of unsuspected cancer in the rectum and often their search is rewarded by finding a growth, perhaps small in extent, but nevertheless unsuspected during life.

Cancer may also occur in the anus, and the terminal portion of the alimentary canal, which is surrounded by the sphincters, known as the anal canal. In this instance cancer may arise either in the mucous membrane of the anal canal or in the skin at the margin of the anus, and is of the squamous-celled type. It is an uncommon disease, more frequently met with in women than in men and excision has been followed by good consequences.

SARCOMATA AND LYMPHOSARCOMATA OF THE INTESTINES.

Sarcomata of the alimentary canal arise in the submucous tissue and may assume the form of a polypus or infiltrate the walls of the canal or project on the surface of the intestine in the form of plaques. All species of sarcomata have been observed. It is also noteworthy that sarcomata attack those regions of the stomach and intestine which are in a measure not invaded by carcinomata. Thus in the stomach sarcomata prefer the body of the stomach and they occur with greater frequency in the small than in the large intestine. In the small intestine the liability to the disease increases from the duodenum to the ileum. Secondary deposits appear to be most common in the liver. An important clinical feature that distinguishes sarcomata of the intestine, large or small, from carcinomata is its occurrence in the early years of life. Many examples have been observed in children. The disease runs a more rapid course, causes more pain, and forms a much larger tumor than is the rule with carcinoma. As a sarcoma often becomes polypoid in consequence of the propulsive action of the bowel, the occurrence of intussusception is a frequent complication. One must be careful in the diagnosing of myomata that are common between the ages of 35 and 55, that these, instead of being typical myomata, are fibrosarcomata or endotheliomata.

Sarcomata of the rectum and anus are rare tumors. They usually cause a bulging in the ischio-rectal fossa, without obstruction. In women the bulging may be on the left or right side of the vagina, perhaps being soft and fluctuating, resembling a large Bartholinian cyst. It is true as Bland-Sutton states "soft, rapidly growing sarcomas in uncommon situations often mimic, in physical signs, inflammatory swellings."[3]

Sarcomata may occur in the subperitoneal connective tissue. These tumors present some peculiar features. In the first place they are nearly always globular and not infrequently resemble a football in shape and size. They have been observed in the neighborhood of the kidney and in some instances this organ occupies a recess in the

Fig. 92.—Annular sarcomata of small intestine. Gut is cut across to show the thickness of the growth. Left half is cut horizontally to show its stenosing of the lumen. Large secondary growth in the mesentery.

tumor. All solid tumors of the mesentery should be examined microscopically. Some of these large tumors are ganglionic neuromata. I have had two instances of sarcomata of the subperitoneal tissue, both of them easily palpable, one resembling a carcinoma of the pancreas and it was not until a radiculitis had occurred manifesting vertebral

involvement that the diagnosis could be made before autopsy, and the second resembling a subperitoneal cyst of large size, in which a differential diagnosis could not be made until death, the patient being too much reduced at the time that I saw him to consider operation.

SYMPTOMS OF NEOPLASMS OF THE INTESTINES.

Benign tumors of the intestine have no symptomatology other than those due to obstruction, either because of occlusion of the lumen of the gut or because of invagination due to traction upon the gut wall causing chronic invagination. Those in the sigmoid may give symptoms of ulceration, diarrhea, and blood and mucus with the stools. When present, there may be much blood passed and small constipated stools, which combination is always suspicious and calls for proctoscopic and X-ray examination. The vast majority of benign tumors of the small and large intestines are not diagnosed during life, since practically all of them are never large enough to cause obstruction or a condition suitable for the development of chronic invagination, and the tumor growths themselves are resistant to the bacteria within the content of the gut, thereby not breaking down in forms of ulceration. Likewise, cancer has no specific symptomatology, and symptoms when they occur are due to obstruction and occasionally ulceration. While it may serve to no purpose practically worth while to differentiate tumors of the small from the large intestine, it is important to remember that the majority of instances of small intestine obstruction are not due to neoplasms, either malignant or benign. The majority of instances of colonic obstruction are due to malignant disease. For this reason an effort will be made to present a symptomatology of obstruction of the small intestine with the hope that it may be of value in differentiating some cases.

Ileus is a term, or name, used to designate as a whole that group of symptoms (colicky pain, constipation or obstipation, distention of the intestine, nausea and vomiting) which indicate the existence of an intestinal obstruction. It is not a disease, not a pathologic entity; it is the expression of a pathologic condition which may vary in degree as it does in its causes. Ileus may be acute or chronic, depending upon the completeness, suddenness and permanency of the obstruction. The chronic variety is for a varying length of time the expression of an incomplete obstruction which under certain conditions may suddenly become complete. The symptoms of acute ileus then supervene. This acute stage may be only temporary or it may be permanent and fatal unless relieved by operation or other procedure.

The principal symptom which first appears in complete obstruction (acute ileus) is a sharp pain in the abdomen, profuse and colicky in character, and of sudden onset. It is a severe pain which soon disappears only to appear again with increased intensity, coming and going with free intervals, there being little or no tenderness on examination. Shortly the eructations of gas begin, the patient becomes anxious and anxiety is shown in the features, while during the colic a cold sweat breaks out. Vomiting starts and continues. All the food or drink is quickly ejected. At this time visible peristalsis may appear, although marked distention is the rule in complete obstruction, visible peristalsis being more common in the incomplete form. However a coil of distended intestine may be seen or may be felt to stiffen under the palpating hand, appearing as an area of increased resistance and giving out a hollow note on percussion. The distention may be entirely local at first but in a few hours it grows more general and the abdomen becomes tense. Enemas may bring away some fecal matter and perhaps a little gas from the bowel, but that happens only very early in the attack and they soon have absolutely no effect. Examination then shows an empty rectum, no gas being passed during the course of the obstruction. There may be little or no rise in temperature and the pulse in the early stage is but little disturbed. As the obstruction continues the pain gradually subsides, the vomitus consisting at first of the stomach content, and then of mucus and bile, finally becomes foul smelling, dark colored and stercoraceous. The features become pinched and sunken, the face pale and slightly cyanotic, the tensely distended abdomen somewhat purplish and absolutely immobile. The pulse then is of low tension and of a rapidly increasing rate. The respiration becomes rapid and shallow, the extremities cold, and the toxemic patient soon passes away. This is the picture of an acute intestinal obstruction, which may be due to a number of causes other than neoplasm. In fact it usually is due to other causes than neoplasm, neoplasms being so infrequently met with in the small intestine.

The distinguishing feature of obstruction of the small intestine is that the pain is more often referred to the region of the umbilicus, the cecum and colon are not ballooned up and the distention is more likely to be central than in the flanks. If the obstruction is very high the amount of distention may be negligible—in fact pain, coprostasis and persistent copious vomiting with a flat abdomen indicates obstruction in the high intestine. Vomiting then is an earlier symptom and becomes stercoraceous more quickly. The patient fails more rapidly, the collapse is more speedy and suppression of urine is more

marked and appears earlier. Indican usually is present and the case as to the locality of the obstruction is not difficult of diagnosis. As the seat of obstruction approaches the large intestine, the distinguishing sign between obstruction in the latter and small intestine is no longer present, and a diagnosis can be made only by X-ray.

Cases of acute ileus must be differentiated from general or spreading peritonitis, acute perforation of gastric or duodenal ulcer or the gall-bladder, acute pancreatic disease, acute cholecystitis, ruptured tubal pregnancy, tabetic crises, torsion of pedicle or ovarian cyst, acute appendicitis simulating acute ileus, thrombosis or embolism of the mesenteric vessels, and renal colic.

The symptoms of incomplete obstruction (chronic ileus) most often met with in neoplasms are usually the forerunner of an acute ileus. The clinical picture is not well defined because a considerable degree of obstruction of the small intestine may exist without giving rise to symptoms so appreciable by the patient as to cause him to seek relief. This is due to the fluidity of the content of the small intestine. The most common symptoms observed are attacks of colicky pain, relieved by the passage of gas and liquid feces. The pain may not be severe in the early stages, but as the condition progresses the pain becomes more intense during the attacks, the patient rolls and tosses and often is in considerable agony. It is its constant recurrence and its practical confinement to a single locality that has significance in the diagnosis of intestinal obstruction. Close observation of the abdomen usually discloses visible relaxation and contraction of a coil or coils of intestine, best seen in thin persons. Heaving waves of intestine are noticed, generally stopping at the point of obstruction. Perhaps the most characteristic symptom is a gurgling metallic sound in the abdomen, often heard by the unaided ear, and frequently noticed by the patient himself. The presence of this symptom is entirely sufficient to warrant a diagnosis of chronic ileus, and of itself justifies the urging of an operation for the removal of the cause. Meteorism is seldom present to any great extent and may be entirely absent. Constipation may not be a noteworthy symptom, and occasionally diarrhea is met with, alternating with the constipation rather confusing the picture. Vomiting and nausea as early symptoms are not important and are uncommon in incomplete obstruction What is a characteristic feature of this type of obstruction is the length of history of attacks of colicky pain, some of these patients in the non-malignant form of obstruction going on from five to ten years.

The most common causes of chronic obstruction are strictures due to tuberculous or malignant disease. Chronic intussusception

CAUSES OF ILEUS (*Bottomley*).

- Ileus
  - 1. Adynamic ileus (paralytic). Symptomatic intestinal obstruction.
    - 1. Operation on mesentery
    - 2. Prolonged strangulation
    - 3. Spinal paralysis from trauma, pathologic lesions, etc.
    - 4. Afferent nerve lesions
    - 5. Reflex
      - 1. Strangulation of omentum
      - 2. Hepatic calculus, colic
      - 3. Renal calculus, colic
      - 4. Torsion of tumor pedicle to degree of strangulation
      - 5. Diaphragmatic pleurisy
      - 6. Acute intoxications (typhoid, pneumonia, etc.)
    - 6. Septic
      - 1. Local peritonitis
      - 2. General peritonitis
      - 3. Embolism (mesenteric)
    - 7. Uremic
    - 8. Tabetic crises
    - 9. Acute pancreatic disease
  - 2. Dynamic ileus. (Excessive muscular action.)
    - 1. Spastic, without known cause
    - 2. Lead poisoning, chronic Tyrotoxicon poisoning
  - 3. Mechanical ileus, from intestinal obstruction.
    - External hernia
      - Inguinal
      - Femoral
      - Umbilical
      - Ventral
      - Lumbar
    - Internal
      - Strangulation
        - Peritoneal and subperitoneal pockets
        - Diaphragmatic hernia
        - Inguinal hernia (internal ring)
        - Umbilical hernia
        - Diverticula
        - Adhesive bands
        - Volvulus
        - Intussusception
      - Obturation
        - Neoplasms
          - Internal
          - External
        - Cicatricial contraction
        - Fecal impaction
        - Foreign bodies (enterolith, round worms)
        - Congenital stenosis

comes next and adhesion of a loop of small intestine to some fixed organ is less frequent.

Cancer of the colon between the ileocecal valve and that portion of the sigmoid which lies above the pelvic brim is the most common cause of definite colonic obstruction and is not uncommonly met with. The vast majority of the cases occur in those between 40 and 70 years of age, although it is not uncommon to meet with cancer of the colon in quite a young person. As a rule in the beginning there are no characteristic symptoms, not only of such as

might be regarded as evidences of predisposing or contributing causes, but of any symptoms by which one might with even reasonable confidence recognize the early stages of cancerous invasion. In a strikingly large proportion of cases the first indication of trouble is an acute intestinal obstruction, these representing about one-third of all which begin with an acute ileus. The general constitutional symptoms like loss of weight, strength, etc. are present, if at all, only in the latter stages.

The presence of a tumor may be made out, although this in my experience is uncommon excepting in the right half of the colon. Intestinal obstruction may be of gradual onset due to a slow narrowing of the lumen of the gut, and the obvious effect of such a narrowing is an increased constipation. Such a condition occurring in an individual of suitable age whose bowels have been in the habit of moving freely and naturally ought in itself to arouse our suspicion. This is particularly true if it is associated with colicky pain, frequent, small and unsatisfactory stools and especially with occasional attacks of diarrhea. The commonest subjective symptoms are frequent recurring colic-like pains with circumscribed tenderness, unpleasant abdominal fullness due to an accumulation of gas, with bowel inactivity alternating sometimes with diarrhea. Particularly the cramp-like pain that increases in severity and frequency until either a tumor can be palpated or the bowel obstruction is complete are, as in the small intestine, the most characteristic features. Vomiting is usually not present until definite obstruction has taken place, then it may be delayed some days. The evidence becomes strong when gurgling of gas through the tumor is heard. The diagnosis is usually certain in the presence of a hard irregular shaped tender tumor. In several cases when the bowel was only partially obstructed, the fecal column was flattened, compressed, stringy and spirally molded, but sometimes the stool was dry and globular. These signs are not of much importance. The evacuations may contain pus, mucus and blood in varying quantity, a hemorrhage being sometimes dangerously profuse. If the neoplasm is sunk into the pelvis, rectal and vaginal exploration may disclose its presence. Neoplasms of the colon in their early development are apt to be round or oval, smooth, and not very large, too small to be palpable. And since in many instances these growths occur in well nourished individuals, no tumor can be made out unless the growth is in the movable part of the colon, this being particularly true in carcinomata of the flexures, especially the splenic. The transverse and descending colon, if palpable at all, are usually freely movable and located below the umbilical line.

Peristaltic waves may be present and when distinct may be regarded as a reliable diagnostic factor.

In intestinal tumors the presence of hemorrhages at a very early period is of great importance. The very small hemorrhages occur quite frequently but a small amount of blood mixed with feces is often overlooked unless searched for.

Cancer of the rectum is the most easily diagnosable of all cancerous conditions of the gastro-intestinal tract. The most common presenting symptom is constipation which Lynch believes is the first and earliest of all symptoms and due to a biologic reaction to the influence of the new growth. The second commonest symptoms are referable to the stomach represented in what might be termed colloquially as indigestion, and probably are the expression, as in the instance of a chronically diseased appendix, of peripheral pathology reflexly affecting the stomach, rendering it hyperesthetic. The symptom of blood or bloody stools, which is usually the first exoteric sign, can occur without ulceration, in which case it may be due to a blocking of the return circulation in the valveless veins leading to the liver. However, most commonly it is due to the ulceration. The next in frequency is the frequent and imperative desire to move the bowels followed by explosive discharges of gas, blood and mucus. This symptom is usually spoken of as the diarrhea of cancer. It is not really a diarrhea in that feces is rarely passed.

The diagnosis here is easily made by proctoscopic examination in which in about half of the instances the tumors are within 7.5 centimeters of the anus and easily within reach of the finger, those higher up requiring a proctoscopic examination.

The symptoms of rectal cancer usually never are initiated with an acute or even a chronic ileus. The symptomatology is entirely local, and since the diagnosis is so easily made by digital examination of the rectum, or preferably proctoscopic examination, no more time need be spent in describing the diagnosis of neoplasm in this situation.

Lastly, in the symptomatology, statement should be made that not infrequently in malignant growths of the stomach and intestine metastases may take place in the nervous system. In the majority of the cases the spinal bones are invaded, more particularly the bones of the vertebra, although they may occur in the soft part of the cord of the brain, peripheral invasion in the form of multiple arthritis not being uncommon in late cancer cases.

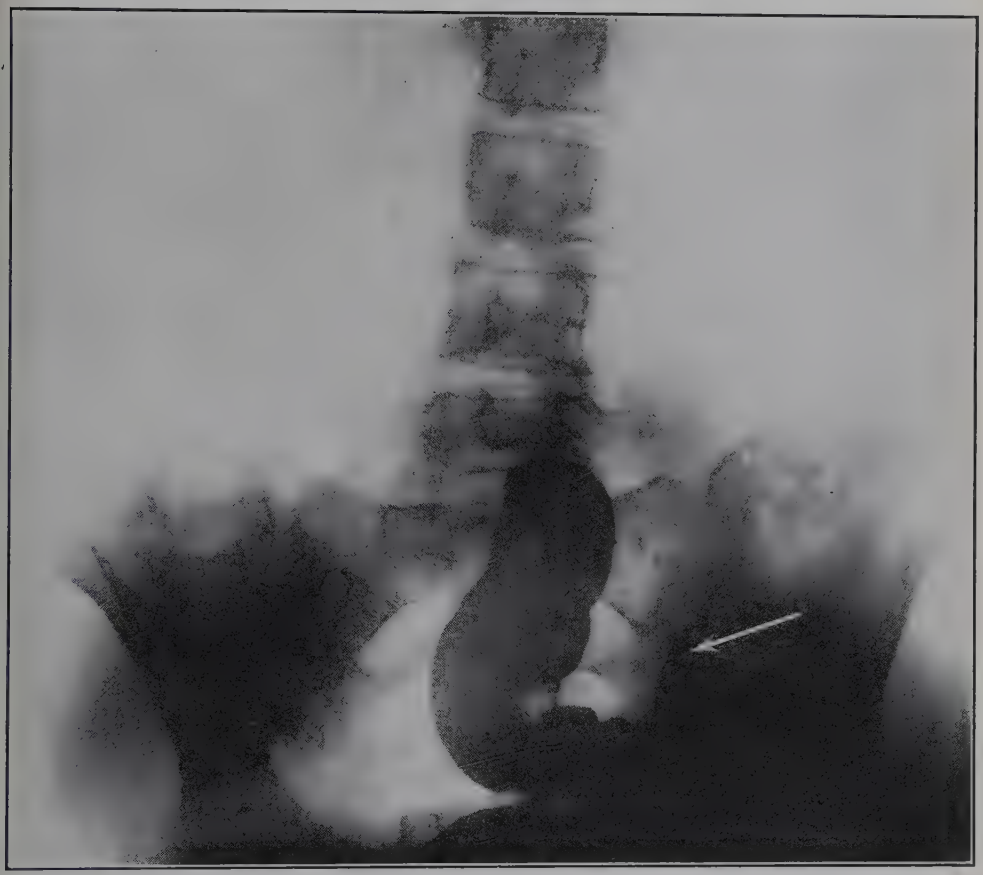
In the diagnosis of carcinomata of the alimentary tract the Roentgen finding of prime importance is a filling-defect. Today the entire alimentary tract is visible Roentgenologically by use of one of the

opaque substances of which barium sulphate is the most popular. In the Roentgenographic examination of the small and large intestine fluoroscopic observation is of the greatest importance. While the X-ray plate method offers much service, careful detail such as the presence of localized pain at a point where the barium mass has hesitated, the grasping of a mass between the fingers to see whether it is movable or immovable, as well as other points, offer detail of

Fig. 93.—Fairly early case of cancer of rectum. (*Case.*)

the greatest importance. The duodenum can be best visualized during examination upon a horizontal fluoroscope. Everything being made ready the liquid food is ingested, the patient lying upon the right side for a few moments. He is then turned upon his back and the examiner places his glove protected hand upon the abdomen and makes pressure over the duodeno-jejunal angle. If the pressure is sufficient there is an incarceration of the barium which occupies the pyloric portion of the stomach and duodenum. Moderate pressure for a few moments will completely fill the duodenum so that its outline can be accurately studied. With carcinoma lower down definite obstruction is met with, and when this exists, a dilated voluminous

PLATE LV



Carcinoma obstructing the pelvic colon. Large amounts of gas in the right and left colon plainly seen. Arrow pointing to site of obstruction. (X-ray by author.)

Carcinoma of the colon. (*Case.*)

PLATE LVII

Carcinoma, annular, obstructing the colon near the splenic flexure. Proximal dilatation of the ascending and transverse colon plainly seen. Arrow pointing to the site of neoplasm. (X-ray by author.)

PLATE LVIII

Carcinoma of the cecum. (*Case.*)

gut may be noted proximal to the stenosis. If the neoplasm occupies or involves the cecum in the neighborhood of the ileocecal valve, there is likely to occur a marked ileal stasis.

There is probably no field of work more satisfactory than diagnosing malignant tumors of the colon early enough for operation, and before very definite obstruction exists. What is true however in this connection is that even with the best qualified Roentgenologists the diagnosis is often missed, due in my opinion to some depending entirely upon the plates, others entirely upon the fluoroscopic examination, or perhaps a fluoroscopic examination not carefully enough made or perhaps not enough time being given to the observation. In carcinomata of the colon the degree of obstruction may be estimated with some degree of accuracy. When the obstruction is almost complete the abdomen is usually markedly distended owing to gas distention of the bowel. My experience agrees with Case that the best method of examination is by means of the barium enema. The barium may be seen to course upward, hesitating or stopping at some site along the way, this generally being the location of the neoplasm, particularly if it can be proven that the gut beyond this contains large quantities of gas. In cases of obstruction beyond the hepatic flexure the cecum and ascending colon may form an extremely dilated sac the thickness of a man's arm, hanging low down into the pelvis, Antiperistalsis alternating with onward peristalsis can be seen in the colon proximal to the lesion, associated with borborygmi. When the obstruction is in the descending, iliac and pelvic colon, and particularly in the iliac colon, the phenomenon of antiperistalsis can be noted also in the left half of the transverse and in the descending colon. The use of the barium with a meal is employed to note where in the transit through the small or large intestine a delay of over a number of hours is met with. This being particularly significant when in addition to the stoppage of the barium meal an accumulation of gas is observed. As I stated before, the enema method of examination is best. Then in cases of carcinomata there is a characteristic arrest of the barium column at the point of hindrance. This arrest may be complete or it may be overcome in a longer or shorter time according to the degree of stenosis. The head of the barium column when it reaches the point of obstruction may present a funnel-shaped shadow. The hindrance to the ascent of the barium stream may be out of all proportion to the amount of actual obstruction. Sometimes if the obstruction is not complete, the barium ascends quickly making further definite detail of observation impossible of accurate conclusion.

TREATMENT.

If the diagnosis of benign or malignant obstruction of the intestine is being made, an operation is in order. It is sufficient to say that it generally is not made until considerable obstruction has taken place, and this being so, nothing can be done in a medical way for the relief or cure of the patient. In the face of a complete intestinal obstruction there is no contraindication to operation, no matter what the age and the condition of the patient (unless he be beyond doubt moribund) and in such an emergency the surgeon is justified in performing whatever operation the condition suggests even if it be the performing of a permanent artificial outlet. But where there is only partial obstruction by an inoperable malignant neoplasm, that is, when the symptoms are obstipation or constipation and diarrhea with the discharge of mucus and blood, and not the alarming and urgent state of complete intestinal blocking, then it is best for the surgeon not to make a colostomy without previous full appreciation by the patient of the nature and the purpose of the operation. In hospital work it is not uncommon to see a patient who has had a colostomy done, slowly dying from a cancer of the bowel, afflicted also with the stench of filth and feces upon his emaciated abdomen and begging for the closure of the outlet which he thought was but temporary. These cases are often found in the homes of the poor and with people who cannot take care of the individual, and I feel that particularly with the poor, the surgeon is not justified in performing a colostomy without full explanation of its consequences.

The ideal procedure is of course, after the colostomy, to do the secondary operation of excision and I have been gratified in a few instances to see most remarkable results accomplished in carcinomata of the colon and rectum by this second-stage operation. It is a well-known fact that no attempt should be made to excise a cancerous growth of the intestine and anastamose the cut ends during the days of acute obstruction, and thus it is that this two-stage operation offers the best result. When an irremovable cancer occupies the cecum, the transverse or descending colon, the loop of the pelvic colon is always available for anastomosis with a suitable coil of ileum. Cancer of the large intestine offers to surgeons a wide field for the exercise of ingenuity and method of making junction and a variety of stitchings have been suggested.

The simplest condition for surgical measures in the colon is a constricting cancer in the middle of the pelvic colon, which admits of wide resection, free removal of the infected lymph-nodes, and the

mobility of the parts facilitating anastomosis. A carcinoma of the anus, anal canal, or lower end of the rectum, suitable for removal, which means that it is not firmly fixed to the sacrum, and has not invaded the prostate, the bladder, or the uterus, may be readily and safely removed. A constricting cancer at the junction of the rectum and pelvic colon has often been successfully excised with the help of an abdominal incision and the cut ends of the bowel successfully sutured end-to-end. The short-circuiting operation in cases of cancerous obstruction of the colon has had a great influence in reducing the number of colostomies. Many instances are known where patients have survived colostomy performed for obstruction due to cancer of the pelvic colon five years or more. This is also true of the anastomosis operation which when possible of being performed does not necessitate an artificial anus. One of my cases of inoperable carcinoma of the ileum in which a coil of normal ileum well above the growth was anastomosed with the fixed part of the ascending colon, lived nine years in perfect health before a return of symptoms in the abdomen, death following secondary deposit in the liver. For details pertaining to these operations the reader is referred to any one of the several books on the subject, the writings of Mayo, Moynihan, and others, the author here simply wishing to reiterate that when definite *bona fide* obstruction of the bowel exists from whatever cause, prompt operation is indicated, the same being true of benign bleeding growths in the lower colon, sigmoid and upper rectum.

References.

[1] Dewis: Boston Medical and Surgical Journal, vol. clv, No. 16, pp. 427-433.
[2] Goodall, Harry W.: Boston Medical and Surgical Journal, Oct. 17, 1912.
[3] Bland-Sutton, Sir John: "Tumours Innocent and Malignant."

CHAPTER XV.

Symptomatic Conditions.

INTESTINAL OBSTRUCTION.
(Acute and Chronic.)

WHAT follows in connection with the above mentioned subject does not present the entire picture of intestinal obstruction. It is essential for the reader to read the foregoing chapter and become acquainted with neoplasm conditions, both benign and malignant, as well as with what follows. When presented with a case of intestinal obstruction distinction as to its cause is not always possible, and in a way the inclusion of the above mentioned condition in the headline of "symptomatic" is somewhat of a distinction without a difference being diagnosable. Very commonly cases of malignant disease present the symptomatology of acute obstruction, although the majority of them are chronic in nature, and it is not always possible to decide that in a case of acute intestinal obstruction the cause is to be found in conditions mentioned in this chapter and that it is not due to a neoplasm. Therefore both sections of the subject should be considered together.

Intestinal obstruction in man, if unrelieved, speedily causes death. The fatal outcome is too rapid to be the result of starvation, and three general theories have been advanced to explain it: (1) A disorder of the nervous mechanism controlling the cardiac and vasomotor systems. (2) A bacterial infection of the organism by the passage outward of bacteria from the intestinal lumen. (3) An intoxication from poisonous substances imprisoned in the intestine orally to the obstruction; the latter being advanced in two forms, the complication being explained as due to noxious substances of bacterial origin in the obstructing bowel, or as the result of an actual toxic secretion by the glandular structures of the alimentary tract.

The advocates of these various theories have done a vast amount of work and experimentation to uphold one or the other, but up to the present time, as is usually the case in medicine when many different reasons are offered to explain something, probably none of them are correct, and therefore the question remains unsettled.

In favor of the first theory, clinical evidence has been adduced. The tachycardia, the low blood-pressure, the profound collapse of the

patient, and the wide dilatation of the splanchnic blood-vessels all point to a loss of activity on the part of the medullary nerve centers. For some time it was believed that this was the result of a reflex disturbance from stimuli acting on the nerve endings in the intestinal wall. But it must be remembered that this train of events may arise from a toxemia or a bacteremia without the intervention of any afferent nerve impulses. Recently, Braun and Boruttau[1] have modified this theory somewhat, and assign the above symptoms to a disturbance of the circulation resulting from interference, chiefly mechanical, with the inherent nerve plexuses in the intestinal wall. It is probable that all of their evidence is indirect and it does not amount to proof of the absence of the toxemia, and this is important in their parallel between the gradual bleeding to death and death in intestinal obstruction, which they claim is a result of failure of absorption. It is true that as death approaches there is a profound disturbance of the nervous control of the heart, blood-vessels, and respiration. But this in all probability is in no sense a reflex disturbance, nor the result of an anemia, *per se,* of the nerve centers.

The infection theory has much more to substantiate it. The intestinal lumen is the normal habitat of innumerable micro-organisms which are potentially pathogenic. When an obstruction exists these no longer have their normal outlet per anum, and conditions favorable for their growth take place. Damage to the intestinal mucosa may allow their passage outward, either into the blood stream, the lymphatics, or directly into the peritoneal cavity. Proof of this is seen clinically in the presence of a peritonitis as a complication of intestinal obstruction without perforation, also in the finding of colon bacillus in the blood as a pathogenic micro-organism. It is probable, as Von Khantz[2] has shown in rabbits in which he produced a low intestinal obstruction, that the bacterial invasion of the blood and the peritoneum takes place. As to which is the first is also a mooted question, some authorities believing that the general circulation becomes affected from the peritoneum, and others that the general circulation can be affected without the peritoneum being concerned in the process. There can be no doubt that a bacterial invasion of both the peritoneum and the blood occurs as the main complication, often a terminal one, but so far as we can tell and can prove by observation, intestinal obstruction will kill with no passage of bacteria beyond their normal place of occurrence in the bowel lumen. If one can judge from experimentation with animals, a clinical examination of the case and also the post-mortem findings, there is such a variety in the findings that a bacterilization does not seem logical. One of course would expect as definitely

in all cases where intestinal obstruction caused death that a local and general dissemination of infection would be met with. Yet such is not the case. Cultures made from the spleen of such animals as are most human not uncommonly show but little, if any, involvement of the peritoneum and liver, and most frequently the spleen cultures are negative. A culture of the femoral blood is likewise often negative, this being true in a few instances of the peritoneum, which is important in connection with the theory when one keeps in mind that cultures from the intestinal lumen always show an abundant growth of various micro-organisms.

The intoxication theory has experimental evidence to support it, and no experiments have proved the absence of a toxemia. Clairmont and Ranzi[3] demonstrated that the stagnated material above an obstruction, after passage through a Reichel or Pukal filter, was exceedingly toxic when injected into the blood of an animal. Kukula[4] found a toxic material in the alcoholic extracts of the intestinal contents in experimental ileus. Roger and Roger[5] and Garnier[6] went farther and proved that the normal content of both the stomach and the intestine are poisonous when thus injected. They did not filter it, but death occurred too early to be the result of an infection. The toxicity was only one-third as great when injected into the portal vein as when injected into a peripheral vein. They also injected it into a mesenteric artery, against the current, so that it was carried backward and then redistributed through branches to a considerable section of the intestine. Here it produced emboli and stopped the absorption, so that large doses were not fatal. This power to cause clotting in the vessels of the intestine is mentioned in this connection because it may account for the gangrene resulting in some of their experiments. Murphy and Vincent[7] found the material from the obstructed or strangulated intestine very poisonous when injected into the peritoneal cavity, and that its poisonous properties were destroyed by boiling or passage through a Berkfeld filter. They concluded, therefore, that living bacteria are the important factors. It does not seem that this conclusion is tenable. Their experiments only prove that living bacteria, if they obtain entrance into the peritoneum, will kill. They do not disprove the presence of other poisons in the obstructed intestine, which, being slowly absorbed over a period of some hours, or days, will kill with the symptoms of ileus. Such poisons may be constantly elaborated and absorbed, but may never be present at any one time in sufficient quantity to cause death when injected into the peritoneal cavity. The symptoms arising from the injection of the intestinal contents into the blood, as reported, differ very markedly from those appearing in the

obstructed animal, and there are many objections as pointed out by Guibe,[8] Braun and Boruttau,[9] Korentchevsky,[10] and others, against accepting these experiments as directly bearing on the cause of death in intestinal obstruction. The lessened absorption from the obstructed intestine is one objection offered. Clairmont and Ranzi[11] found that potassium iodide placed in the bowel above the obstruction, was excreted from the kidneys in greater quantities than normal up to eight or ten hours after the obstruction was produced, the excretion rapidly decreased after that and came to a stop in about fifteen hours. Braun and Boruttau[12] gave strychnine to their obstructed animals, and from the resulting symptoms they concluded that absorption became markedly reduced from the start. On this experiment they base their chief objection to the intoxication theory.

Hartwell and Houget have undertaken to prove in their experiments that the symptom development may be very slow and death retarded, if proper precautions are taken in excluding damage to the intestinal wall so that there is only a minimum absorption of poison. They believe that a more important objection than the lessened absorption, to using the evidence of the injection experiments as a proof of the intoxication theory, is that many substances injected directly into the circulation are poisonous, whereas, if they have to pass through living cells to get into the blood, their poisonous properties are entirely destroyed. Peptone is such a substance, and is a good illustration in this connection. The injection of peptone into the blood produces symptoms of poisoning, which may end in death. The absorption of peptone from the intestine is, on the contrary, a normal physiological process. Therefore, the mere presence of substances in the obstructed intestinal tract, which act as poison when injected into the blood, is no indication that these substances cause the death resulting from an obstruction, since they are normally found in the intestine in conditions of continued health. To accept the view that such a death results from a toxemia it must be conceded either that new poisons are elaborated and absorbed or that an abnormal absorption of the poisons normally present takes place. In their experimentation they came to the conclusion that the essential factor causing the symptoms and death in intestinal obstruction does not lie in the poisons *per se,* but in the production of lesions which favor their abnormal absorption.

In searching for the source of the poison three possibilities are met with: (1) Foodstuffs or substances derived from them; (2) true bacterial toxins; (3) secretory substances from the alimentary tract and digestive glands, or their derivatives. It was readily conceivable

that any one of these factors may be present. Throwing out of consideration the bacterial theory, Hartwell and Houget said the essential point in their experimentation was injury to the normal mucosa. We know that a high obstruction produces much more severe symptoms, and is more rapidly fatal, than a low obstruction. The bacteria, however, are much more numerous in the lower bowel than in the upper. Roger and Garnier[13] found the most toxic substance in the duodenum; the least in the colon. The toxicity, too, was greater in unobstructed conditions, than in the obstructed conditions, and it grew less the longer the obstruction continued while McClure[14] has shown that the bacteria increase rapidly when an obstruction is produced. Vidal[15] found that the toxicity could be decreased by injecting simultaneously with the duodenal contents glycerin extract from the jejunal mucosa lower down, and this cannot be explained on the ground that bacteria are the exciting cause of the poison. V. Baracz[16] found that dogs with a double occlusion of the lower ileum and cecum might live for many weeks. At the end of this time only a moderate quantity of material was found in the loop. The contents were rich in bacteria. The intestinal wall showed marked changes; most in the form of a hypertrophy, but sometimes an atrophy was present. The microscopic findings are not given. In one case the animal was killed after four hundred and twenty-five days while in a condition of perfect health. Here the bowel loop, about 20 centimeters in length, was found enormously distended, filled with 365 grams of a foul-smelling, greenish material, which was rich in bacteria; a bacillus, the Bacterium aërogenes and a leptothrix being isolated in cultures.

These cases seem to point to the conclusion that the stagnation of bacteria and their toxins in the intestine is not sufficient to cause the symptoms seen in intestinal obstruction. Indeed, they, with the facts cited above, seem to make it doubtful whether micro-organisms play any essential part, either directly or indirectly, in producing these symptoms. This question, however, must remain an open one for the present, and the writers can offer no positive answer to it. There still remain the stagnated foodstuffs, and the stagnated glandular secretions of the stomach, intestine, liver, and pancreas as the source of the poisonous materials. Draper,[17] of Rochester, following Maury of New York, has worked on the theory that the duodenum secretes a substance or substances which normally are rendered non-toxic by passing over the jejunal mucous membrane lower down. When obstruction is present the duodenal substances are not brought into contact with the antibodies, and, hence, remain poisonous and cause death. Vidal earlier advanced this view. Draper, to support this theory, has

done some elaborate experiments in the way of sidetracking the various secretions, but his reports are somewhat contradictory, and he himself says they are very inconclusive. Recently he has reported that feeding dogs which have an obstruction just distal to the duodenum with the excised mucous membrane from the jejunum and ileum of other dogs, prolongs the life of the obstructed dogs. The difference between the control dogs and the fed dogs, however, was not marked, and the exceptions where a fed dog died in a shorter time, or one not fed lived a longer time, were too numerous to make the total result at all convincing.

Whipple,[18] who has been prominent in the study of this problem, has advanced the theory that the intoxication of intestinal obstruction and closed intestinal loops is due to the absorption from the intestinal mucosa of a definite proteose. He believes that the evidence for the intervention of bacteria in the situation is not at all conclusive, whereas there can be no question that the intestinal mucosa is essential to the production of the poison. A recent contribution[19] stimulates interest anew, and is likely to provoke further critical discussion of the question. In confirmation of the results of previous investigators, it has been found that dogs with an isolated closed loop of duodenum or jejunum die in from forty-eight to ninety-six hours, in most cases with perforation of the isolated loop and general peritonitis. But there is usually no excessive vomiting and hence no fatal dehydration of the body tissues. The dehydration factor has been made responsible by some of the investigators for the severe symptoms of intestinal obstruction. When the isolated and closed loop of the jejunum is sterile, complete occlusion of the blood-vessels to the isolated loop has no effect on the dog; but if the loop is not sterile, the occlusion of the circulation in the loop causes death in from twenty-four to forty-eight hours with the usual symptoms of complete intestinal obstruction.

Hartwell and Houget concluded from the results of their extensive experimentation with animals the following:

"1. A high intestinal obstruction, that is, 10 to 30 centimeters from the pylorus, in dogs, may not produce death for ten days, provided the gut wall is not damaged. If it is damaged by section and inversion the average life is only half as long.

"2. There are found in the kidney and liver cellular changes which are the same as those found in many toxic diseases. The intestinal mucosa is found to be damaged to such an extent that it may readily be conceived that it has been deprived of its natural defence against the passage of toxic substances, unaltered, through it.

"3. Bacterial invasion of the blood and organs does not necessarily occur.

"4. Dogs deprived of food for forty-eight to seventy-two hours, may die as early as those fed ten to twenty hours before the obstruction is produced. Decomposition of foodstuffs is not, therefore, an essential element in causing death.

"5. If a double occlusion of the alimentary tract, with re-establishment of the continuity of its lumen, above the lower ileum be produced, the damage to all the tissues is greater than with a simple obstruction, and the course of the disease to a fatal termination is shorter. A double occlusion in the lower ileum produces much less damage than in the upper.

"6. The action of the gastric juice, bile, pancreatic juice, and duodenal secretions are not a requisite in producing the symptoms and pathological changes seen in intestinal obstruction, because these are produced by a double occlusion in the upper ileum when none of these secretions are blocked.

"7. Simple occlusion of the pylorus does not necessarily produce any evidence of a toxemia in two weeks, and the gastric mucosa at the end of this time shows no evidence of being damaged.

"8. The above findings indicate that death from intestinal obstruction in dogs results from the presence of toxic substances in the circulating blood which produce fatal lesions in the kidney, liver, and other tissues. The essential factor which admits these substances into the blood is an injury to the lining cells of the intestine caused by the irritating action of the stagnated contents, together possibly, with the mechanical damage due to stretching. The poisons themselves may arise from the secretory activity of the various digestive glands, or from bacterial activity. They may be the same as those found in the normal tract or they may be substances newly formed under the conditions of stagnation. Whatever their source, they are innocuous so long as the mucosa remains normal."

An abundant series of experimental observations has seemed to the Chicago physiologists to warrant the following conclusions: Closed intestinal loops in which the bacteria are first removed are not incompatible with life. Closed intestinal loops in which bacteria are present but in which tissue necrosis is prevented are not incompatible with life. Closed aseptic intestinal loops in which the blood supply is completely shut off are not incompatible with life. The normal secretions of the duodenum and jejunum are not toxic when allowed to drain into the abdominal cavity. These results do not support the theory of Draper of a normal toxic secretion of the duo-

denal mucosa, neutralized by the jejunal mucosa, or the perverted secretion theory of Whipple. In obstructed loops of bowel, occlusion of the circulation by distention from accumulated secretion may easily arise. Necrosis follows, and bacterial toxemia completes the story. Thus the Chicago investigators conclude that bacterial activity plus the necrotic tissue or the result of the action of bacteria on necrotic tissue are the important factors in the rapid death in simple closed intestinal loops.

Intestinal obstruction may be caused by strangulation, intussusception, twists and knots, strictures, tumors, by abnormal content and by paralysis of the muscular coat of the bowel. Benign and malignant growths and intussusception due to them (invagination) is treated in the foregoing chapter, together with tumors causing intestinal obstruction. Paralysis of the muscular coat of the bowel (dynamic ileus) is treated in the succeeding chapter.

Etiology and Pathology: *Strangulation.*—This is the most frequent cause of acute obstruction and in the cases analyzed by Fitz, occurred in 34 per cent. of 295 cases, and in those of Leichtenstern 35 per cent. of 1134 cases. The most numerous of causes are adhesions, and in the analysis of Fitz's table, they follow in numbers in this order: vitelline remains, adherent appendix, mesenteric and omental slits, peritoneal pouches and openings, and adherent tube. The bands and adhesions result, in a majority of cases, from former peritonitis, and are seen commonly following operations upon the pelvic organs in women. The strangulation may be recent and due to adhesion of the bowel to the abdominal wound or a coil may be caught between the pedicle of a tumor and the pelvic wall. Late occlusion after recovery from the operation is due to bands and adhesions. Such adhesions may follow operations for appendicitis, gall-bladder disease and gastroenterostomy for ulcer or from the pelvic operation. Bands and adhesions are often seen following unoperated upon cases of appendicitis, tuberculous peritonitis, and a twist affecting the small bowel. In some instances a history of a trauma many years back is obtained, this no doubt accounting for the production of adhesive bands. Such adhesions may be present on the left side, for reasons the cause of which is not very plain, but probably due to long standing constipation which has brought on the production of ileopectineal adhesions, these cases often leading to acute volvulus or to recurring attacks of abdominal pain, or to definite attacks of partial obstruction.

Adhesions causing intestinal obstruction are met with at any point of the intestinal canal. Such adhesions may be met with in the second part of the duodenum onward, and not uncommonly one finds

an adhesion case in which the site of the adhesion is in the first loop of the jejunum. I have seen several cases of this sort, and in each instance the adhesive band forms a tight bridge across the lumen of the gut, sometimes also twisting the gut upon itself, the contortion being from left to right and about half a turn. In the small bowel there are three locations in which constrictions and adhesions are most likely to occur. They are the first portion of the duodenum, the terminal portion of the duodenum, and at the terminal portion of the ileum. Instances of duodenal adhesions in the gall-bladder region are numerous. Such adhesions may bridge over to involve the hepatic flexure of the colon. Most times these adhesions are slight, without causing throughout life intestinal obstruction. In fact the majority of the cases found are of that nature, but they may be met with quite firm in character and be the sole cause of an acute as well as a chronic intestinal obstruction. One not uncommonly sees a retraction of the duodenum and pylorus upward and to the right, and at times constriction and dilatation of segments of the duodenum.

The adhesions to which attention should first be directed are those found on the posterior surface of the cecum and the posterolateral wall of the ascending colon. In some cases these may be perhaps better designated by the term agglutination than adhesions, as the peritoneal layer of the bowel seems to be simply stuck to the peritoneum of the parietal wall without there being any visible organized band connecting the two. If the bowel be raised up, a distinct white line frequently becomes visible showing the edge of the agglutinated surfaces, and on further traction the parietal peritoneum becomes drawn in. A variety of adhesions are found in distinct bands of newformed tissue extending from the cecum or the ascending colon to the parietal peritoneum to the appendix, to the omentum, or to the lower end of the ileum. These are all undoubtedly of inflammatory origin and may vary much in number and thickness, and frequently have a direction upward and backward from the bowel to the postero-lateral parietal wall.

Constrictions in the terminal portion of the ileum are rather hard to explain excepting that they are caused by diseased appendices. Pain and kinks may cause a modified form of constriction, although in the majority of instances it does not seriously interfere with the passage through the gut.

Constrictions of the colon are most liable to occur at the hepatic, splenic and sigmoid flexures, although they may occur anywhere. Such constrictions are more likely to be extensive and to be associated with folds of the bowel, although they may be rather isolated, pro-

ducing no definite kinks and with no actual obliteration of the lumen. It is probable that colonic strictures are secondary to low grade colonic inflammation due to bacterial conditions within the colon wall causing a peritonitis and the development of adhesions in that way. As stated, adhesions of the left side of the large bowel are commonly the cause of volvulus, probably due to the rising of the bowel to overcome the adhesion and stricture state and in that way the proximal coil become displaced with torsion.

Richter's Hernia may be found at all ages but usually at the extreme of life. It is defined as abdominal hernia in which a portion of the circumference of the bowel is imprisoned, reducing, but not entirely obliterating, a portion of the intestine. It is more common in females and is apparently limited to adults. The lower limb is the portion usually engaged. In some cases the lumen may permit fecal passage. The symptoms are unreliable and misleading and present little uniformity. The gravity of the case depends upon the condition of the bowel, and there are no symptoms to serve as an index of intestinal viability. In about one-third of the cases the symptoms are typical of this condition. In the remainder, those of a mild incomplete obstruction. In one-tenth of the less severe cases there are bowel movements on the first or second day. The vomiting is of a mild and intermittent type, fecal vomiting being present in about 12 per cent. of the cases. Should the bowel become gangrenous from the stricture usually fecal vomiting does not occur. It must be remembered, however, that in Richter's hernia complete obstruction is not unusual. Constipation is a consistent symptom. Diarrhea, however, has been present and is a bad omen. Obstipation is the general rule. Distention of the abdomen is not marked, as these constrictions are water-tight before they are air-tight. The clinical diagnosis is difficult and has been made in about 50 per cent. of cases, confirmation of the diagnosis being in the first presentation of an irreducible hernia inflamed or strangulated. The condition simulates a small incarcerated omental hernia. The mortality is high, probably as Treves put it at 62.2 per cent.

PERICOLONIC MEMBRANE.

The cecum and its near relatives and coadjutors, the ascending and transverse colon, have received considerable attention from surgeons, internists and Roentgenologists in connection with certain membranous structures which are frequently found enveloping these organs in whole or in part, and are designated by such terms as

pericolic membrane, membranous pericolitis, pariepocolic membrane, and Jackson's membrane. The American profession has contributed largely to the elucidation of this subject which for a long time had been noted at *post mortem*, and Jackson[20] gave it a definite clinical

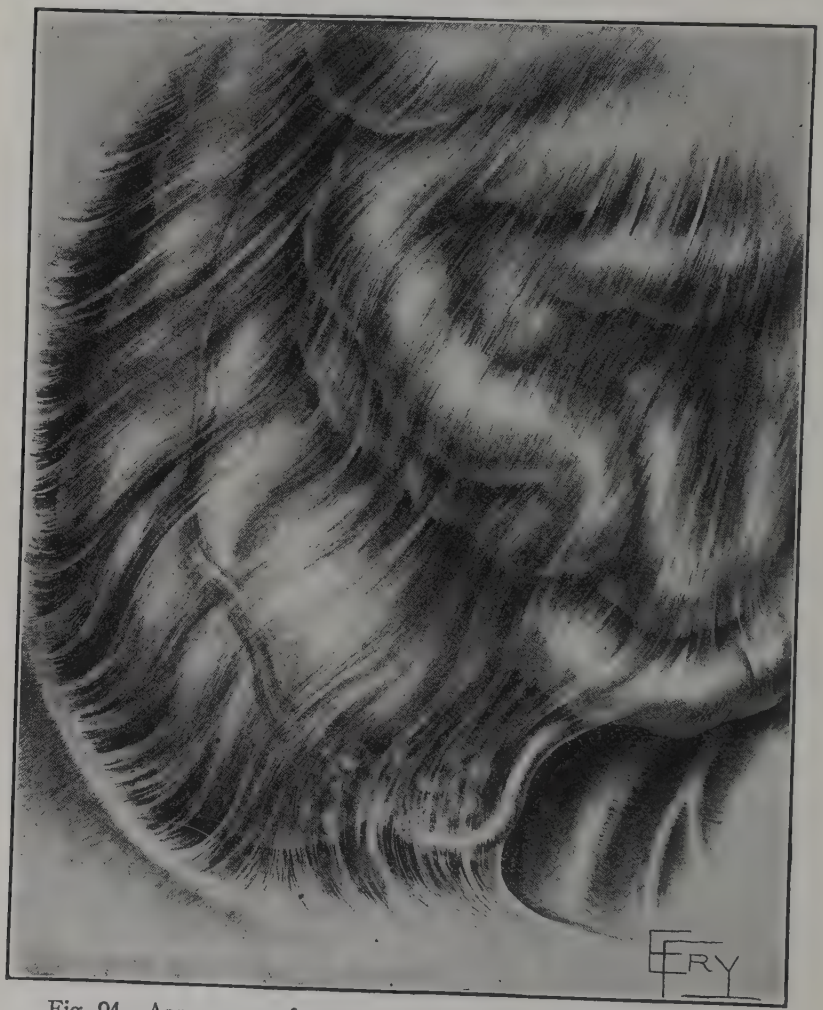


Fig. 94.—Appearance of extreme involvement of ascending colon, appendix and small intestine by newly formed pericolic fibers (drawn directly at operation). (*Pilcher.*)

entity with a symptom complex and plan of treatment. Jackson's paper with its account of the pathology is probably the best classic for these conditions. There are numerous writers here and abroad who have contributed important papers.

There is still some question as to whether peritoneal irritation can cause the formation of these membranes. The author takes the position that membranes so formed are of the tissue type and do not represent the congenital type spoken of here. The evidence of a congenital origin of Jackson's membrane is almost indisputable. The thin diaphanous veil springing from the postero-lateral abdominal

Fig. 95.—Abdomen of a newborn infant showing a Jackson's membrane covering the ascending colon. The rudimentary omentum may be seen at the free border of the transverse colon, unrelated to the membrane. (*Cheever.*)

wall on the right, overlying in varying degrees the ascending colon but so loosely attached to it that it may be divided and lifted up with scarcely any bleeding, leaving an apparently normal colonic peritoneal surface, and the absence of any thickening or hyperplasia, or change in the consistency of the colonic wall, constitutes a picture different from an inflammatory process. Such membranes are met with in fetal

life and a study of the new born infant at *post mortem.* While Jackson claims to have had no observation of pericolonic membrane in infancy or childhood, I have often met with them in post-mortem work. Cheever[21] in an examination of thirty still-born infants ranging approximately from six months to the full term, in the anatomic labor-

Fig. 96.—Same as Fig. 95. Cecum of fetal type and appendix raised to show a genitomesenteric fold connecting the mesentery of the terminal ileum with an undescended testicle. The small intestine has been removed except the terminal ileum, whence may be seen springing a Meckel's diverticulum. *A,* Undescended testicle. *B,* Meckel's diverticulum. *C,* Cecum. (*Cheever.*)

atory of the Harvard Medical School, met with four instances of characteristic veil-like membrane extending from the side over the hepatic flexure and ascending colon. He believed, and rightly, that the membranes are developmental and that their origin was in the

rotation and descent of the cecum from its first point of fixation beneath the liver.

This membrane may be seen by the ascending colon presenting the appearance of being thickened or edematous and on close inspection the visceral peritoneum seems to be raised from the bowel wall by reason of the presence of a subendothelial hyaline-looking substance or exudate. The endothelial layer of the peritoneum is slightly movable on the bowel wall, gradually becomes separated from the bowel as a distinct layer, and beneath it and covering the bowel wall there is formed another layer of endothelial cells. A raised, thin, transparent membrane remains attached to the bowel at the longitudinal band at which point small blood-vessels may be seen entering and spreading over the thin membrane. In older cases the membrane is thicker, particularly along the line of the blood-vessels, which also become larger and more numerous. It is probable that in some instances eventually the membrane becomes intimately attached to the ascending colon, and it is probable that it is in these cases that some of them justify operative procedure, because I have seen a colon puckered up so as to be practically obliterated on the external surface of the bowel. The membrane is usually found in the upper two-thirds of the colon, the cecum being free. The upper end is almost invariably at the end of the hepatic flexure and the lower at from 1 to 1½ or 2 inches above the lower end of the cecum.

Symptoms.—In by far the largest number of individuals who have these membranes no symptoms of them are present, and when they are, usually other pathologies met with in the right abdomen are responsible rather than the presence of the membrane itself. However there are some individuals in whom the membrane is seen to cause symptoms, whether because of fixation of the appendix or because of confinement of the colon interfering with its peristalsis. My experience is that it is but rare that the colon is enough confined by the membrane to produce symptoms, although it is probable that, insofar as the appendix is concerned, their production is possible.

When symptoms are due to the membrane, they may be described as a discomfort, increasing at times to positive pain in the right iliac region, in which location there is usually more or less of an indefinite sense of tenderness on pressure. This tenderness may be increased by matters of diet or exercise, occasionally taking on a colicky character suggestive of paroxysms of muscular spasm of the bowel. In a few instances the paroxysms of discomfort with tenderness on pressure, take place at the time of the mass movement of the right colon to the transverse and splenic flexure. Ofttimes there is a

gaseous distention of the right side present at the time of the distress, and there may in a few instances be a loss of weight and the usual symptom complex of auto-intoxication and neurasthenia. Obstinate gastric symptoms are not infrequently to be added. More or less cecal stasis is present in cases due to defective peristalsis, a more or less obstruction due to a diminution of the bowel lumen by the veil perhaps having a conflicting band, enterospasm, and the pathology in the mucous membrane which comes on from a long standing auto-intoxication in which there is destruction of the cellular elements.

Treatment.—For quite some years there has been a surgical leaning to the proper method of treating these cases. Not a few have had their appendices removed, even the gall-bladder operated upon, of course without benefit. I might go further now and say that most unusual is the case of operation in which Jackson's membrane having been removed that it has in any way benefited as far as the symptoms are concerned, and it is a fine question in most every case in which the symptoms cannot be ascribed to anything other than the membrane, whether operation should be performed or not. I must admit that whereas formerly I was surgically inclined, today I am most conservative. My position is to temporize with these people in medical ways unless there is definite proof that what is tantamount to obstruction exists. However, after the institution of a proper diet (anti-constipation), one which is arranged according to the type of toxemia present, together with such medical means as rest, tonics, the building up of the general tissues of the body, etc., if no definite benefit is derived or such is not sustained, it might be wise to make an exploratory incision, perhaps to do some surgery upon the membrane. I would again give the caution that in at least 10 per cent. of individuals this membrane is more or less present, and that in but very few of these are the symptoms due to the membrane itself. Usually it is due to recurrence of some acute appendicitis, duodenal ulcer, inflamed Meckel's diverticulum, intestinal toxemia, etc., rather than to the membrane or constrictions, or anchorage caused by it. However, in an obstructive case in which no pathology is met with, it may be wise to divide the membrane, preferably with a cautery, by the method advised by Hofmeister, which Jackson claims in some instances has prevented their reforming. One should however be a little guarded in prognosis as to cure, for these people represent a type in which the best results from surgery are not generally obtained.

Meckel's Diverticulum has been found at autopsy in 2 per cent. of human beings. Although extensive statistics on this point are

wanting it is apparently much more frequent in the male sex. It is a congenital anomaly due to subinvolution of the omphalomesenteric canal. The attachment of the diverticulum usually is in the distal half of the ileum and is within three feet of the ileocecal valve in the majority of cases. It arises from the convex border of the intestine, infrequently from a more lateral position, and rarely between the two leaflets of the mesentery. It varies in length up to 10 inches, the average being 2 or 3 inches. In shape it may be conical, with the base directed toward or away from the intestine, globular, or, most frequently, cylindrical. Rarely it presents a bifid appearance at the tip, the result of a secondary protrusion through a defect in the muscular coat. Its diameter is approximately that of the ileum, but may be larger or smaller. As a rule it is not provided with a mesodiverticulum. It is never multiple, and is composed of the same structures as the four coats of the intestine from which it arises. Its mucosa is provided with villi, Lieberkühn's glands, and Peyer's patches. Incomplete involution of the umbilicomesenteric vessels is frequently indicated by the presence of a fibrous core extending from the diverticulum to the umbilicus, or to the adjacent mesentery or small intestine. Perverted involution occasionally leads to an associated stenosis or atresia of the adjacent ileum.

In the absence of complications, Meckel's diverticulum does not give rise to any symptoms, but a slight hint as to its presence in a few instances may be afforded, according to Gray, by deep retraction or scarring of the umbilicus. The most common lesions by which it manifests itself clinically in the order of their frequency are, intestinal obstruction, diverticulum open at the umbilicus, diverticulitis, and hernia. It is the cause of intestinal obstruction in 6 per cent. of the cases. Commonly the diverticulum, congenitally or secondarily attached by its tip, performs the rôle of a band in causing trouble. In other instances obstruction was produced by an intussusception beginning with the diverticulum, by a volvulus or torsion of the intestine, by a rent in the mesodiverticulum, by congenital stenosis or cicatricial stricture of the ileum, or by a free diverticulum wrapped firmly around one or more loops of intestine.

After an analysis of 69 cases Halstead found that intestinal obstruction due to Meckel's diverticulum was suggested by: "(1) Onset in childhood or early adult life. (2) A history of preceding minor attacks. (3) Inverted cone-shaped configuration of abdomen indicative of obstruction in the upper small intestine. Absence of distention in the flanks was conspicuous during the early hours of the attack. (4) Local meteorism and occasionally visible peristalsis es-

pecially below the right costal arch. (5) Fecal vomiting, as a rule early, but this is denied by Cazin, Gray, and Forque and Riche. (6) Tenderness in the right side on a level with or just below the umbilicus, or according to Berard, great sensitiveness of the umbilical region when the diverticulum is attached to the umbilicus. (7) Association of other congenital malformations is very exceptional, and was present in only one (harelip) of the 69 cases."

Gray notes that the early localized meteorism may be situated below the level of the umbilicus rather than above when the strangulated intestinal loop has passed under the diverticulum from above downward. As further confirmatory evidence might be added male sex, and a history negative of inflammatory trouble adequate to cause adhesions or bands. Gray has made a special study of invagination of Meckel's diverticulum and has been able to collect forty cases. He found that after invagination the inverted diverticulum may cause an immediate intestinal intussusception, may hang free in the intestine causing recurrent hemorrhages, or may form an intestinal polyp after fusion of its serous surfaces. The invaginated diverticulum alone was found in 7, a secondary intussusception of the intestine in 22, and an independent intussusception in 1. Twenty-three were males and 7 females. The preponderance of males seemed to be in excess of the normal sex variation and probably was due to their greater physical activity. This affection occurs most commonly in childhood and early adult life. Only two patients were under two years of age. Two-thirds of the cases presented an antecedent history suggestive of mechanical or inflammatory lesions, as evidenced by attacks of colicky abdominal pain with or without blood in the stools. The symptoms caused by the invagination of the diverticulum alone or in association with intestinal intussusception were those of a more or less complete intestinal obstruction and were not characteristic. The average duration of the attacks was three and one-half days, but in 12 it was over five days. In general the symptoms were mild the first one or two days and then became rapidly worse. Pain was always severe, usually violent, and commonly situated in the umbilical region. Vomiting was a constant symptom. Of 25 cases, 8 passed blood by rectum and 17 did not. Constipation was seldom absolute. A palpable tumor was noted in 16 cases. Tenderness was absent prior to the onset of peritonitis. General abdominal distention was present in some cases, and absent in others. Of 6 patients not operated upon, 4 died, and 1 recovered after sloughing and spontaneous evacuation of the intussusception. There was performed intestinal resection in 15 cases with 8 deaths, resection of Meckel's diverticulum

alone in 5 cases with 1 death, and enteroanastomosis to relieve obstruction in 2 cases, both of which proved fatal.

Forque and Riche collected 88 cases in which the diverticulum was open at the umbilicus. Seventy-five of the patients were males and 9 females, and 62 were infants under 1 year of age. The diverticulum is commonly patulous throughout, forming a congenital umbilical fecal fistula, but rarely it may be impervious at some part of its length, forming a mucous fistula of the umbilicus which can be differentiated from fistula of the urachus by the intestinal type of its mucosa and by the probe passing backward rather than downward along the linea alba. The fistula may close spontaneously or may persist more or less intermittently for years. Through the umbilical opening there may occur prolapse of the mucous membrane only, of the diverticulum alone, or of the diverticulum and the intestine. Treatment should consist of excision of the diverticulum. If not undertaken at once because of the patient's age, its potential dangers demand its removal at a later date, even though the fistula has closed. In recent prolapse with undamaged intestine, laparotomy with reduction of the prolapse and excision of the diverticulum is indicated. In prolapse of long standing adhesions between the serous surfaces of the gut often prevent reduction and require resection of the intestine. Prolapse complicated by gangrene necessitates intestinal resection, and, depending upon the patient's condition, and immediate or subsequent intestinal anastomosis.

The inflammatory affections of Meckel's diverticulum are analogous to those of the appendix. Pathologically, diverticulitis may assume the catarrhal, interstitial, or gangrenous form. Ulceration may be non-specific or result from typhoid fever, tuberculosis, or actinomycosis. As many as 13 perforations have been found in one instance (Makins). A correct preoperative diagnosis of diverticulitis has never been reported. The symptoms of diverticulitis closely resemble those of the corresponding form of appendicitis. As a rule the localization of symptoms in diverticulitis is somewhat nearer to the umbilicus than to McBurney's point, but it is by no means sufficiently characteristic to exclude appendicitis. In very rare instances umbilical scars, marked retraction of the navel, or an antecedent history of umbilical fecal fistula might be a determining factor against appendicitis. An acute inflammatory attack simulating appendicitis in an individual from whom the appendix has been removed, and in whom there is no other ascertainable cause for the symptoms, favors a diagnosis of diverticulitis. This contingency has not occurred in any of the recorded cases, but it seems only a matter of time before it will arise.

Of 59 cases of diverticulitis collected by Forque and Riche, 40 were males and 12 females, while in 7 the sex was not specified. Of 41 patients operated upon, 15 died, 21 recovered, and in 4 the result is not given.

Meckel's diverticulum was found in an inguinal or femoral hernia in 52 of the cases collected by Forque and Riche, and in upward of 60 cases by Gray. The latter also found reports on 3 instances of obturator hernia. Porter found 10 umbilical hernias in his collection of 184 cases of abdominal crisis caused by Meckel's diverticulum. Bunts directs attention to an anatomical predisposition to hernia of Meckel's diverticulum, in that the lower ileum to which it is attached has a mesentery longer than any other part of the intestines.

Ekehorn explains some of the cases of congenital inguinal hernia on the basis of adhesions forming during fetal life between the diverticulum and the testis or the adjacent peritoneum. With the descent of the latter into the scrotum the diverticulum is drawn into the open processes vaginalis. Complete reduction of a hernia of Meckel's diverticulum is rarely possible. The characteristic shape of the diverticulum occasionally may be recognized on palpation in thin patients. In strangulation affecting the diverticulum alone the symptoms of intestinal obstruction are wanting, the local symptoms predominate and resemble those of inflamed hernia, while the constitutional symptoms appear late and are mild unless peritonitis supervenes from perforation of the diverticulum. The details of many of the cases were so scanty that Gray excludes all but 42 from his classification. Of these, 30 were inguinal and 12 femoral. Of the inguinal hernias, 26 were males and 3 females, and of the femoral, 4 males and 8 females. The hernia was on the right side in 19 inguinal and 4 femoral, and on the left in 7 inguinal and 4 femoral. The patients came under observation at all ages, but most commonly during adult life or middle age. In many the diverticulum had remained quiescent in the sac for years. Strangulation was noted in 27, incarceration or inflammation in 2, and irreducibility in 11. In 17 of the 27 cases of strangulation the diverticulum alone was found in the sac. All of the patients were adults. In 14 the hernia had existed for a period varying from six months to thirty-three years, and in all of these extensive inflammatory adhesions existed between the diverticulum and the sac. In 3 cases the hernia had not been observed prior to the onset of strangulation. In 2 of these the diverticulum was not adherent. Only 7 of the patients exhibited any signs of strangulation, and in most of them there was some complicating factor to account for the symptoms. In 3 fatal cases there was a coexistent stenosis of the ileum

leading to perforation of the ileum in 2, and acute obstruction in 1. Two patients died following perforation at the base of the diverticulum. In 7 of the 17 patients there was perforation of the diverticulum with the formation of an external fecal fistula.

In 10 of the 27 cases of strangulation, either intestine or omentum, or both, was found in the sac in addition to the diverticulum. In many the strangulation involved only the intestine. In 2 the diverticulum was perforated. These patients were all infants or children, and only 5 recovered. Of the 11 cases of irreducible hernia, 10 were males and 1 female. Four were adults, 4 children, and 3 infants. The diverticulum was adherent to the sac in 8 cases. Ten patients were operated upon, the diverticulum being resected in 7 and returned to the abdomen in 3 without a single death.

INTUSSUSCEPTION.

In this condition one portion of the intestine slips into an adjacent portion, forming an invagination or intussusception. The two portions make a cylindrical tumor, which varies in length from a half inch to a foot or more. As the course of the intussusception is onward, an upper section of the gut invaginates into a lower, and as the process proceeds, the inner and middle layers increase at the expense of the outer layer. An intussusception really consists of three layers of bowel; the outermost, known as the intussuscipiens, or receiving layer; a middle or returning layer; and the innermost or entering layer. In Fitz's statistics of intestinal obstruction they represented about one-third of the cases of acute obstruction. It happens about twice as often in males as in females, and is most common in early life, 34 per cent. in infants under 1 year and 56 per cent. under the tenth year. Wiggin reports that of 103 cases in children, nearly 50 per cent. occurred in the fourth, fifth and sixth months.

The site of the invagination varies, and any designation pertaining to an individual case is merely to give it an anatomical name. When the ileocecal valve descends into the colon it is known as ileocecal, and this form occurs in 75 per cent. of the cases. Sometimes the invagination is so complete as to present the ileocecal valve in the rectum. When the lower part of the ileum passes through the ileocecal valve it is known as ileocolic. The ileal is that form in which the ileum alone is involved. The colic is confined to the large intestine, and when the colon and rectum are involved it is known as the colico-rectal.

Irregular peristalsis is the essential cause of intussusception. Invagination may follow any limited, sudden, and severe peristalsis.

After a short time peritonitis ensues, or a marked acute injection of the serous membrane. The portion of the bowel affected is usually large and thick, and forms an elongated tumor with a curved outline. The parts are swollen and congested owing to the constriction of the mesentery between the layers. The entire mass may be of a deep livid-red color. The constriction of the mesenteric vessels is not so complete, or in the early stages of a case there is only congestion, and perhaps a thin layer of lymph. This condition of intussusception can be reduced but when it has lasted for a few days, lymph is thrown out, the layers are glued together and the entering portion of the gut cannot be withdrawn.

The anatomical condition accounts for the presence of the tumor, which exists in two-thirds of all cases; and the engorgement, which results from the compression of the mesenteric vessels, explains the frequent occurrence of blood in the discharges, which has so important a diagnostic value. If the patient survives, necrosis and sloughing of the invaginated portions may occur, and, if union has taken place between the inner and outer layers, the caliber of the gut may be restored and a cure effected in this way. Not a few cases of this kind are on record.

Intestinal obstruction may result from congenital strictures, although these cases are exceedingly rare. Much more commonly the condition is that of complete occlusion, either forming the imperforate anus or the congenital defect by which the duodenum is not united to the pylorus. These cases of course will be met with very early in life, a few days after birth, and when stenosis is situated somewhere above the anus, gradually a vomiting ensues which in a few hours becomes constant, the infant vomiting all that is taken into the stomach. The abdomen distends and the general condition of the little patient soon becomes alarming. As a rule no meconium is voided.

Intestinal obstruction may be caused by stenosis from a cicatrix, resulting from ulceration, tuberculosis or syphilis, more rarely from dysentery, and seldom from typhoid ulceration. These cases, of course, produce evidences of chronic obstruction, as will be described, or that of an acute obstruction in which it may be possible to obtain the history of chronic obstruction which has gone on unobserved by the patient.

There are other causes of intestinal obstruction, such as compression and traction. Tumors of neighboring organs, particularly of the pelvic viscera, made by pressure causing occlusion, or by adhesion formation and traction. The healing of tuberculous peritonitis may cause contraction and narrowing of the coils of the gut.

Reports are on record of foreign bodies, such as fruit stones, coins, pins, etc. causing intestinal obstruction. It is a good rule, however, to remember that anything which can pass through the esophagus is small enough to go through the gastro-intestinal tract. Medicines, such as magnesia or bismuth, have been known to accumulate in the bowels and produce obstruction, but in the great majority of cases here the condition is caused by feces, gall-stones or enteroliths. Obstruction by gall-stones is not infrequent, these, of course, occurring more frequently in women than in men, and usually in about the fiftieth year of life.

Symptoms.—Whatever the underlying cause, acute obstruction when established gives rise to a certain series of symptoms. They vary in severity according to the nature of the obstruction, and the other circumstances of the patient's illness. Treves has advanced the point that the obstruction itself is not the important thing giving rise to the symptom complex—the manifestations of the lesion do not depend primarily upon it. Rather they are evidences of severe injury to the peritoneum, and correspond to an acute peritonitis. After this manifests itself, we have the symptoms of the obstruction itself, and finally if this be unrelieved, the terminal symptoms of intestinal sepsis or auto-intoxication supervene. It is because the symptoms of peritonitis are the primary ones that occasionally a difficulty in diagnosis arises at the very beginning of acute obstruction. The case becomes more clear when the signs of occlusion or sepsis come on, but then it is often too late to follow a diagnosis by successful treatment. While the picture of an acute intestinal obstruction that has been standing for some days is distinctive and unmistakable, in the first stage it may simulate quite a few of the other acute intra-abdominal lesions.

The subjective symptoms of intestinal obstruction are pain, nausea and vomiting, and retention of gas and feces. The pain of acute intestinal obstruction comes on suddenly, and is generally referred to the neighborhood of the umbilicus. It may come on while the patient is walking, or more commonly, during the performance of some action. It is described as being much like a severe form of colic, but subsequently becomes continuous and very intense. Vomiting follows quickly, and is a common and most distressing symptom. At first the contents of the stomach are voided, and then greenish, bile-stained material, and soon, in cases of acute and permanent obstruction, the material vomited is a brownish-black liquid, with a distinctly fecal odor. This sequence of gastric, bilious, and finally, stercoraceous vomiting is perhaps the

most important diagnostic feature of acute obstruction. The vomiting does not relieve the pain, nor is the pain relieved by pressure. Vomiting is more often seen in obstruction of the small than of the large intestine, and this is because the lower the obstruction, the more space there is for intestinal content to occupy, and thus the less the vomiting, although in a length of time (somewhat longer than obstruction of the small intestine), in cases of obstruction of the colon, frequent vomiting will occur.

The constipation may be absolute, without the discharge of either feces or gas. Very often the contents of the bowel below the stricture are discharged, or may be removed by enemata, or other artificial means. Therefore we must not be deceived by the expulsion of feces or a small amount of gas following the administration of an enema. In intussusception we often have a spurious diarrhea, at times bloody at the onset of the disease. It is apt to be deceptive, especially in those cases in which a gastroenteritis with a pronounced diarrhea has preceded the intussusception. Tenesmus is at times associated with the constipation, almost exclusively in intussusception and cases where the obstruction is not quite complete. Distention of the abdomen usually occurs and when the large bowel is involved, it is extreme. On the other hand, if the obstruction is high up in the small intestine there may be very slight tympany. In this tympany, the abdomen which at first is not very painful, becomes acutely tender.

The constitutional symptoms of the onset are severe. Collapse occurs in some cases owing to the acuteness of the pain and the suddenness of the occlusion. I agree with Deaver that I have not found it to be a marked feature until the obstruction has been established for some hours at least. The temperature may fall or rise slightly, although when peritonitis occurs it rises. If the peritoneal irritation is so great as to give rise to true shock, the pulse may show this by being rapid and weak. Often, however, in cases of acute obstruction the pulse may be accelerated and become suggestive in character of severe peritoneal irritation, or a beginning peritonitis. As the case goes on the patient becomes pallid and anxious and finally collapse symptoms supervene. The eyes become sunken, the features pinched and the skin is covered with a cold clammy sweat. The amount of urine excreted is diminished if the obstruction continues, the tongue is dry and parched, and the thirst is incessant. The urine is high-colored, scanty, and there may be suppression, particularly when the obstruction is high in the bowel. This is probably due to the constant vomiting of the

small amount of fluid which is absorbed. As a rule a very high leucocytosis is present. In such an extreme case, the patient usually dies from shock or sinks into coma sometimes in from three to six days.

Increased peristalsis is invariably seen in acute intestinal obstruction. It can be recognized in several ways. In a thin subject we can at times see the exaggerated peristaltic movement through the abdominal wall; often by the application of the ear or the stethoscope to the abdomen the peristaltic movements are audible. Exaggerated peristalsis is said to be generally more marked when the obstruction is comparatively low down in the intestine. This has been my observation because it stands to reason that if the obstruction is high (in the small intestine) there is not enough gut above it to throw the depth of an abdomen into tension enough so as to see visible peristalsis on the anterior wall. The best examples of peristalsis in intestinal obstruction that I have seen have been in those where the obstruction is from the lower end of the ileum to the splenic flexure of the colon.

The symptoms of chronic obstruction are sometimes observed in the back history of cases of acute obstruction, and sometimes they may be prominent enough to make a diagnosis of chronic obstruction before the acute condition ensues. As a rule there is a history of long-standing constipation; there may be a history of the discharge of mucus, or in some instances, fecal masses which have been channeled so as to allow the contents of the upper portion of the bowel to pass through. This in elderly people is not infrequent. On the other hand, there may be the story of small movements having a frequency of being repeated with an absence of a sense of entirety after each movement, or the movements may be ribbon-shaped or very small in caliber, seen in obstruction in the lower end of the intestinal canal. Vomiting, pain in the abdomen, gradual distention, the whole condition subsiding and then returning again are not uncommon findings in the history.

On the other hand, not uncommonly in stricture whether cicatricial or cancerous, the symptoms of obstruction are very diverse. What makes it difficult is the intermittency of the symptoms. Constipation may occur which gradually disappears, and attacks of abdominal distress, or accumulations of gas which finally disappear. Usually the obstruction is not enough to cause vomiting, alhough I have seen several cases of chronic obstruction in which the nausea was very marked at the time the obstruction was active, subsiding when transit through the obstructive area had been accomplished,

and in others, the presence of vomiting at these times. The vomiting, however, is not of a biliary or fecal character. Usually the emptying of the content of the stomach suffices, the attack culminating about that time. As a rule, the general state of health is not seriously impaired, the patient, however, very slowly and gradually becoming anemic and emaciated. I have seen many cases of advanced carcinoma of the gut in which there was no very apparent effect upon the general state of health, although most of them will admit to a degree of weakness that is progressive. When one remembers all that has been brought out in connection with malignant disease of the intestine, namely, the history of attacks of pain in the abdomen, not very sharp in character, these being accompanied by distention, distress, gas, perhaps some nausea and vomiting, all of which subside when the bowels have been moved, which is accomplished with greater or lesser difficulty, depending upon the type of the obstruction, we have them *in toto*. These symptoms are intermittent, sometimes the space between them being several years. When such a history is obtained, search for some cause of chronic intestinal obstruction is in order. The reason that it is important is because it is just this type of case that terminates some day in an attack in which the obstruction is complete, the symptomatology then being that of acute intestinal obstruction.

Of course in the chronic type of case the Roentgenographic examination of the abdomen is very valuable. This is not so much worth while nor practical in the acute case, because here we deal with a very short run in which the symptoms rapidly intensify and the condition is almost self-evident. But in the chronic case the stoppage of the barium bolus for hours at a time at some point in the canal, particularly when in the portion of the gut where the barium is accumulated there is the presence of a considerable amount of gas, intestinal obstruction is to be strongly suspected.

Diagnosis.—Acute pancreatitis, renal and biliary colic at times give us symptoms simulating the beginning of an obstruction, but usually they show their true nature in a very short time. Hernia must be excluded which is by no means always easy as fatal obstruction may occur from the involvement of a very limited portion of the gut in the external ring or in the obturator foramen. A thorough rectal, and in women a vaginal examination, should be made which will give important information as to the condition of the pelvic and rectal contents, particularly valuable in cases of intussusception in which the descending bowel can sometimes be felt. In cases of obstruction high up, the empty coils sink into the pelvis and perhaps can be

detected. In the inspection of the abdomen there are important indications; there are the special prominence in certain regions, the occurrence of well-defined masses, and the presence of distended coils in active peristalsis. In obstruction of the lower end of the large intestine not only may the horseshoe of the colon stand out plainly when the bowel is in rigid spasm, but even the pouches of the gut may be seen. When the cecum or lower end of the ileum is obstructed the bulging is in the lower central region, and, during the spasm, the coils of the small bowel may stand out prominently, one above the other, either obliquely or transversely placed—the so-called "ladder pattern." In obstruction of the duodenum or jejunum there may only be slight distention of the upper part of the abdomen, associated usually with rapid collapse and anuria. Acute pancreatitis at its onset may simulate acute intestinal obstruction. The location of the pain and its persistence in the epigastrium, the more marked collapse with perhaps slight jaundice, and the rapid pulse would make us think of a pancreatitis. The tympanitis is also chiefly in the upper abdomen, and the vomiting as a rule is very persistent.

Poison and cholera have been mistaken for obstruction, but rarely so, and are usually easily distinguishable.

Appendicitis, beginning as it does by pain and vomiting, may at times simulate obstruction. The absence of complete constipation, the early vomiting, combined with localized tenderness and spasm in the region of the appendix will put us on the right track.

Cases of ovarian cyst twisted on its pedicle may present the symptoms of an acute obstruction. Here as in other conditions for abdominal diagnosis, the rectum may be inflated by an atomizing bulb fastened to a rectal tube, jets of air being sent in one after the other, distending the sigmoid region, and the descending, transverse and ascending colon, and if in a few minutes after the introduction of air into the rectum, the air is present in the cecal region—in such an instance no obstruction in the colon exists. But in the absence of an insufficiency of the ileocecal valve, no air can be introduced into the small intestine, and even if it could be, the position of the small intestine is so close to the center of the abdomen, that it is not possible to deduct accurately enough to make this examination worth while in strictured conditions of the small intestine.

Practically no other conditions give rise to the symptoms liable to be confused with those of acute intestinal obstruction. The diagnosis of an obstruction of some sort is then, as a rule, not usually difficult except in a few cases where much morphia has been given.

Nature of the Obstruction.—The differential diagnosis of the various forms of primary intestinal obstruction is most unsatisfactory. The probable differentiation on the basis of age, etc., intussusception which gives peculiar symptoms, and volvulus which may be relieved by enemata, are the only ones easily differentiated.

Strangulation is not common in very early life, and in a number of instances, there have been previous attacks of abdominal pain, operation, or history of an old peritonitis, to give one a clue. Neither the onset nor the character of the pain gives any information. A tumor is not common in strangulation, and is only present in about one-fifth of the cases. Fever, likewise, is not of diagnostic value.

Intussusception is an affection of childhood, and is of all forms of internal obstruction the one most easily diagnosed. The presence of tumor, bloody stools, and tenesmus are the important factors. The tumor is usually sausage-shaped and felt in the region of the transverse colon. It becomes evident on the first day in more than one-third of the cases, and is practically always found on the third day. The blood may be mixed with mucus; the tenesmus may be distinct, although fecal vomiting is not common. Abdominal tympany is a symptom of slight importance, occurring in only one-third of the cases. Volvulus is diagnosable when it can be reduced, by enema or by inflation. When it cannot be, or when the obstruction is due to some other cause, it is rarely diagnosed. However, the frequency with which it involves the sigmoid flexure is to be borne in mind. The air inflation of the rectum method is often here a valuable aid in differential diagnosis.

In fecal obstruction the condition is usually clear, as the feces can be felt per rectum and also in the distended colon. Fecal vomiting, tympany, abdominal pain, nausea and vomiting are late, and are not so constant. In obstruction by gall-stones a few of the cases give a previous history of gall-stone colic. Of late years many instances have been reported in which peritonitis following disease of the appendix has been mistaken for acute obstruction. The intense vomiting, the general tympany and abdominal tenderness, and, in some instances, the suddenness of the onset are very deceptive. It must be remembered that in appendicular disease the presence of a high leucocyte count would argue in favor of some acute inflammatory trouble as against obstruction, particularly if this count were high in the polynuclears.

In hospital work one not uncommonly meets with cases that have been operated upon which in time develop symptoms of intestinal obstruction. These are divided by Deaver into three forms, 1. Those

following immediately after operation; 2. Those resulting from mechanical intestinal obstruction; 3. Those the result of septic peritonitis. The following of Deaver's is the best exposition that I have read, and is true to clinical application.[22] "The obstruction following immediately after operation is in most cases due to excessive handling of the viscera, or at times apparently caused merely by prolonged anesthesia and operation. Such an obstruction might occur with equal frequency in clean and septic cases. It is a true form of dynamic ileus, produced directly by the causes mentioned. Most important of all the factors is, as Cannon and Murphy have shown experimentally, the question of handling. Their researches show that in animals while prolonged anesthesia alone and simple exposure of the gut to the air, had no very great effect on peristalsis, even slight handling of the stomach and intestines caused very marked gastro-intestinal paresis. This occurred even when the viscera were handled intra-abdominally or under saline solutions. When they were removed from the abdomen and gently handled the effects were more marked, and an extreme degree of paresis resulted from rough handling of the intestines. It has seemed to me at times that simple traction by retractors carried out too forcibly, and causing irritation of the parietal peritoneum, has caused some intestinal paresis, other causative factors being apparently absent. And not alone by mere digital or instrumental handling can the viscera and parietal peritoneum be injured. The rough or careless introduction of gauze packs can give us the same bad results, as can also the inexpert placing of materials for drainage. It is not enough to be aseptic within the abdominal cavity, we must also be gentle. Surgeons are prone to think and act as if with asepsis anything is permissible within the abdominal cavity, but this is to my mind an entirely wrong attitude. Whatever may be the true reason of a paresis following manipulation. whether it proceeds directly from some purely local cause or indirectly from the spinal cord, its occurrence is an undoubted fact.

"Post-operative obstruction due to mechanical causes occurs generally in septic cases. It may be due to newly formed adhesions or to gluing together of coils of bowel, or bowel and omentum by plastic exudate. Naturally this is rare in clean cases. The greater the area infected, the more it is disturbed, and the more roughly it is invaded, the greater will be the likelihood of an obstruction. Insufficient drainage of septic abdominal fluids, pus and sero-pus, would lead to circumstances favoring its occurrence. This is but another point showing us the importance of good drainage in any case where drainage is indicated. Much of the drainage, especially where gauze

is used, defeats its own intention, and keeps in septic material which it should carry off. Then again, drainage gauze if applied so as to irritate normally or abnormally contiguous gut surfaces, will also tend to give rise to conditions favoring the occurrence of obstruction. I have known gauze drains to obstruct directly the intestinal lumen, so that their removal alone caused all symptoms of obstruction to subside. In this possible mechanical obstruction by gauze and its insufficiency for the drainage of amounts of fluids and its tendency to irritation, we have the strongest argument for the use of tube drainage, when drainage is really indicated. It is surer and quicker and less liable to pave the way for complications.

"Post-operative obstruction as the result of sepsis is not uncommon—indeed it is more common than both the other forms. The spread of a peritonitis or beginning of one after operation, seems at times to be unavoidable. And in the presence of a marked peritonitis at time of operation, one which is widespread and virulent, it seems often to be impossible to avoid obstruction. Naturally, the more the infection is spread during the operation, the greater is our liability to produce a general peritonitis and its sequel—peritonitic obstruction. And after it subsides, it leaves the patient in danger of having an obstruction occur at some future time by adhesions or bands formed during the course of the peritonitis.

"It is often a matter of difficulty to determine which of the three factors mentioned is the cause of a post-operative bowel paresis. At first, *i.e.*, immediately after operation, we may be in doubt as to whether we have a simple paresis or one due to a beginning peritonitis —later on, whether it is due to a local peritonitis, the result of a forming secondary collection or of some mechanical cause. Often two or more factors are associated."

Between obstruction due to paretic distention or obstruction due to bands, kinks and so forth, diagnosis is usually clear, particularly if the patient is seen early in the course of the disease. A diagnosis from obstruction due to peritonitis consequent upon the formation of a secondary collection of pus, on the surface would look as though it should be readily and easily made, yet this is not always true. Obstruction due to embolism and thrombosis of the vessels of the mesentery is a diagnosis difficult and often impossible to make.

Mechanical ileus has a delayed invasion, from a few days to several weeks after operation. The onset is sudden, the pain is sharp and localized; tenderness and meteorism are confined at first to one region, vomiting may not come on at once but is conspicuous as the case progresses.

In peritonitis the invasion is early, pain, restlessness and anxiety are observed from the first; vomiting is one of the first symptoms; meteorism is general; epigastric distention is first noticed; the pulse is fast, it may be wiry; the temperature is elevated; constipation may not be absolute at first or even later on.

Treatment.—When intestinal obstruction exists purgatives should not be given, and no morphine should be given until the abdominal diagnosis is reasonably clear and a course of procedure definitely decided upon. In the presence of strangulation from whatever cause immediate operation is indicated. The same rule holds good in intussusception, although in a few cases which have been seen early the intussusception has been reduced by mechanical manipulation, likewise inflation of air into the rectum has reduced some. Of course it must be remembered that those that are reducible are only a few in which the diagnosis was made in the first few hours. When the output of lymph has taken place and the intestinal walls are glued together, no method of medical treatment is of any value and operation is definitely indicated. The same is indicated in those in which it may be judged that the obstruction is due to twists, (not impaction kinks as tumors), and strictures due to cicatrices, and compression and traction from tumors of neighboring organs. Where it can be judged that the obstruction is due to an abnormal content, conservatism would be in order, and here purgation with castor oil or enema may be worthy of a trial. Great care must be taken in the post-operative cases, and differentiation between definite obstruction due to mechanical means and that due to paresis of the bowel wall, or peritonitis is to be made. In my experience most of the cases that have been operated upon for intestinal obstruction shortly following abdominal operation have been mistakes in diagnosis on the part of the surgeon. For a surgeon to operate upon a case of post-operative dilatation not due to mechanical reasons often means the destruction of the individual, while to do so in that due to peritonitis caused by the first operation is practically to encourage the same thing. While the point of differentiation here is sometimes very difficult, as a rule a careful study of the case and some medical treatment clears up the doubts. When it is a case of ileus, the washing out of the stomach, and best of all the use of the trans-intestinal lavage method with a hypertonic solution, is usually most helpful. Irrigation of the large bowel with injections should be practised, the amount allowed to flow in from a fountain syringe being carefully estimated. The more difficult differentiation is between acute intestinal obstruction due to mechanical means shortly following operation, and peritonitis. In

peritonitis the pain is usually general in the abdomen, intense and aggravated by movement and pressure. Sometimes the patient takes a position to relieve the tension of the abdominal muscles. The greatest pain is below the umbilicus, but if it is in the upper part of the stomach it may be referred to the back, the chest or the shoulders. The respiration is superficial, costal in type and it is painful to use the diaphragm. Generally the abdomen becomes distended, tense, and tympanitic on percussion. The pulse is soon affected and has a peculiar wiry character. There may be no temperature because the patient may be in a prolonged condition of shock following the operation, but I have always felt that if there is any temperature, and it is common to meet with it, peritonitis should be suspected. I believe, too, that in the differentiation, surgeons do not pay enough attention to what our forefathers drew attention to in connection with peritonitis—namely, the Hippocratic facies—"a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the color of the whole face being brown, black, livid, or lead-colored." It must be remembered in peritonitis, the tympany is usually excessive, owing to the great relaxation of the walls of the intestine by inflammation and exudation. Splenic dullness may be obliterated, liver dullness likewise, although this is a very late symptom. As a rule these cases do not live long enough for effusion of fluid, although in my hospital work I have met with it in enough cases following operation where intestinal obstruction was believed to be the cause, to look for it every time, and when present to definitely diagnose a peritonitis instead of intestinal obstruction.

For the tympanitis, turpentine stupes and hot applications may be applied. In cases of chronic obstruction the diet must be carefully regulated, opium and belladonna being given for paroxysmal pains, the bowels kept free, and abdominal massage persisted in. The main thing here is to find the cause. It may be situated in the changed bacteriology of the gut or an infective process somewhere else in the body, which when possible should be removed, or relieved so that it is less of a causative factor of adhesions. In some of my cases of chronic excessive intestinal toxemia in which definite adhesions of the gut were present, the cure of the condition of the toxemia caused a resorption of the adhesions. This I have proven to myself in a positive way.

Lastly it should be mentioned that if the obstruction is acute, or becomes complete, resort must be had to surgical measures, but care should be taken in the so-called chronic obstruction cases, all of which

PLATE LIX

Chronic small intestine obstruction in a case of carcinoma of the ileocecal region. (*Case.*)

are partial, that very careful diagnostic work is done and that the judgment arrived at should be along conservative lines. I mean by that persisting with medical measures rather than promptly resorting to operation.

INTESTINAL OBSTRUCTION DUE TO MOTOR PARALYSIS.
(Adynamic or Paralytic Ileus).

As has been mentioned, the cases after operation which present the manifestation of intestinal obstruction are most commonly those due to peritonitis, less frequently those due to motor paralysis of the gastro-intestinal canal, and least common of all those due to bands, adhesions, twists, volvulus, etc. caused by the manipulation of operation or incident infection. While the latter requires operation, most of the former are made distinctly worse by it, and of these intestinal obstruction due to motor paralysis requires mention here.

Every operator of experience in abdominal surgery has met with the distressing post-operative condition of abdominal distention, the degree of which may range anywhere from a mere accumulation of gas in the intestine causing only slight distress, to a well developed condition of ileus with impending death.

A review of the literature on this subject shows that volumes have been written, and that the term ileus used without qualification leads to endless confusion, since it designates only a syndrome and describes no definite morbid condition. The late John B. Murphy published a classification of ileus and endeavored to give a definite meaning to the term, retaining the term ileus and carrying with it this meaning: "Ileus is a complexus of symptoms occurring usually in a fairly regular form and order, and represented by pain, nausea and vomiting, meteorismus and caprotatus." He divided ileus at that time into two classes—dynamic, meaning obstruction and excessive power of the bowel, and adynamic, meaning a paralytic ileus or absence of power in the bowel.

Experimental investigations into the influences of the innervation of intestinal movements have proven uncertain, but Pflüger established by a series of investigations the following facts: The vagus nerve innervates the entire small intestine and irritation of its fibers either causes movement throughout the small intestine or increases peristalsis. Irritation of the splanchnic nerve on the other hand tends to inhibit movements of this portion of the intestinal tract, and also causes anemia of its blood-vessels, while the division of these same nerves causes a hyperemia.

Cannon and Murphy[23] undertook to settle the question as to the location of dynamic ileus in the way as to whether it was of central or peripheral origin. They showed that when the nervous connection between the alimentary canal and the central nervous system was intact, as in asthenia of animals afflicted with distemper for example, food would lie in the stomach or intestine all day without the slightest sign of a peristaltic wave pushing over it. There is a total stoppage of the motor activity of the digestive organs. When, however, the spinal cord and brain are disconnected so that the stomach and small intestine are entirely deprived of their extrinsic innervation these organs have been observed exhibiting their normal activities although the animal was to the last degree exhausted and toneless. In one instance an animal very limp and apathetic after the spinal cord and vein had been disconnected, was fed at intervals of an hour for five hours in which the Roentgen ray revealed normal peristalsis of the stomach and normal motor activity of the small intestine at each observation, although at the end of the time the animal was moribund, gastric peristalsis still being active. They state that if the nervous connection between the brain and spinal cord had been intact in this case the persistence of the activity of the alimentary canal, according to their experience would not have been seen, concluding that the general bodily condition would have affected the canal unfavorably through the nervous system.

Attention is drawn to the fact that the splanchnics may be acted upon reflexly, in that way slowing up the motor power in the stomach and small intestine. In injury, for instance, this is probably a characteristic manifestation, although in animals injured in which the alimentary shock could be done away with, or very much minimized, providing an anesthesia and operation were not added, there was simply a retardation but not a complete paralysis. Cannon and Murphy came to the conclusion that not only could ileus paralyticus be produced without mechanical change in or about the intestinal wall, but that the condition is one of inhibition of innervation conveyed to the stomach and intestine through the splanchnic nerves. Alvarez has shown that there are zones in the gastro-intestinal tract from which there is initiated a peristaltic wave, the wave passing from one zone to the next nodal point from which they are enhanced. This power of the intestinal canal is resident in the Meissner and Auerbach plexuses, and that would explain the motor ability of the intestine when these are disconnected from the brain and spinal cord, and also from the splanchnic from which they no doubt obtain most of the innervation which has to do with peristalsis.

Surgeons are aware of the fact that etherization, even when considerably prolonged, does not seem to cause a delay to any marked degree of the discharge of food from the stomach, the same being true of exposure of the gut to the air. An unusual cooling of the gut likewise seems to have no effect. But experience has shown that when the stomach and intestines are handled even gently, no movements of the stomach are seen and no discharge into the intestine takes place. In Cannon and Murphy's observation stripping the gut between the fingers resulted in increased inertia of the stomach and extreme sluggishness of the small intestine, in one case no food having left the stomach at the end of seven hours, and in another case the food had not reached the large intestine twenty-four hours after the feeding. Manipulation of the stomach and intestines, therefore, even gently, and under the most favorable circumstances, produces much greater effect in the direction of post-operative inactivity than any other of the factors under control during operation. The so-called "gas pains" following laparotomy are due to this condition.

It is therefore clear that in any case of dynamic ileus a distinction must be made between the inactivity due to inhibitory impulses of the central nervous system and inactivity due to local disturbances in the gastro-intestinal wall. Cannon believed that this distinction permitted the following of several courses in considering the possibility of treatment. As has already been shown the normal stomach and intestine are capable of carrying on their motor activities quite independent of the central nervous system. Consequently, if the inhibition is extrinsic any agent that would check the delivery of an inhibitory impulse from the cord to the canal would permit the canal to resume its normal functioning. If on the other hand, the inactivity is the direct result of local disturbance this same agent would have no effect in promoting the restoration of peristalsis. In this connection it is pointed out that when the inhibition is imposed from the spinal cord, it is possible to start the movements of the canal by depressing the action of the cord or by blocking the impulses from the splanchnics by salicylate of physostigmine which for a long time has been used in post-operative ileus. Experimental action shows that physostigmine is of no benefit unless the canal is stagnant, and then it is only temporary. Cannon and Murphy showed that the administration of the drug at the second hour had produced suddenly a very abundant discharge from the stomach into the intestines, and that the intestinal peristalsis was also very active, but that in the fourth hour and thereafter the stimulated motility had quieted down. They rather concluded that temporarily at least salicylate of physos-

tigmine will destroy reflex inhibition of the alimentary canal and cause the normal movements to proceed.

It must therefore be concluded that post-operative ileus can to some degree be brought about reflexly by an effect upon the splanchnic as upon the gastro-intestinal tract by disturbance of innervation from the brain and spinal cord, and while exposure and prolonged anesthesia seem to have little or no effect experimentally, the practice shows that they are somewhat of a feature when the handling of the part particularly when outside of the abdominal cavity is a factor, and that rough handling of the part, such as stripping the intestine or drawing strongly upon the mesentery, causes a sectional paralysis for some hours of time. Lastly it must be mentioned that there are cases of post-operative ileus following operations performed elsewhere on the body than the abdomen.

Symptoms and Diagnosis.—In a general way, three fourths of all cases follow operation on patients between 10 and 40 years; the younger or older the patient, the less the danger. Most of the cases follow abdominal operation; males and females are about equally affected.

Definite gastric symptoms may be most acute and characteristic or may be absent. Naturally, the more severe the dilatation, the more likely will these be present; still, markedly dilated stomach in serious cases may exist without vomiting, pain, tenderness, thirst or scanty urine, all of which are characteristic symptoms. The onset of the attack when severe is generally sudden. The patient survives an operation and the post-operative course appears normal for a few days, or possibly as long as two weeks afterward. Suddenly a distention of the stomach takes place accompanied by profuse and persistent vomiting of large amounts of fluid, pain in the epigastric and umbilical regions, which may be steady or colicky, tenderness in the stomach region, and symptoms of collapse. Muscular rigidity is usually absent, arguing against a peritonitis. The abdomen swells, due to the enlargement of the stomach, the right hypochondrium becomes prominent and the left flattened, and the general gastric tympany on the upper left side shows an enlarged organ, the lower border being below the umbilicus, possibly extending almost to the pelvis. The transverse measurement of the organ is also increased. On the passing of a stomach-tube to relieve the gaseous distention and lavage to wash out the fluid, the size of the organ quickly diminishes. The stomach may be so distended with gas that the succussion sounds may not be distinguishable. Visible peristaltic waves may occur, but they are rare in the severe cases, being more common in the moderate grades

that have existed untreated for several days. In such an event, following an operation or trauma, a stomach-tube should be passed at once, and the return from the stomach examined. It can then be seen that an unaccountably large collection of fluid is present in the stomach, this at first being quite yellow, then yellowish green or green when regurgitation from the upper part of the small intestine is present, and finally brown with solid particles and a fecal odor when the condition has existed for some hours.

A treacherous severe type may exist in which the characteristic symptoms are absent or are too slight and indistinct for definite diagnostic purpose. These are seen in the post-operative cases in which anorexia and gastric fermentation or intestinal flatulency have been present more or less continuously. For no assignable reason, suddenly, a quick change is noted in the appearance and general condition of the patient. The countenance becomes dusky or pallid. The face appears "pinched", the pulse more rapid, smaller and thready in character, the temperature drops and may even become subnormal, the hands and feet cold, and the abdomen more or less distended. Such cases may not present much on examination of the abdomen, although the right upper aspect may be slightly prominent. As vomiting may not occur, and stagnation of the stomach always exists, dependence must be placed upon the use of the stomach-tube for diagnosis, and fortunately this method of examination is usually conclusive, although its use in lavage of the organ is not so often curative as in the first form. These cases are an alarmingly fatal type, death occurring within a few days.

The most favorable cases are those of the milder types, in which the paralysis may be said to be incomplete. These comprise varying grades and are about evenly divided between the post-operative and mixed cases, such as occur in typhoid fever, pneumonia, etc. In these the onset may be slower and more indistinct, and the abdominal examination virtually negative. Noting the history of cause and remembering that the possibility of this condition in any degree always exists following operation, the diagnosis is suggested by a sudden unfavorable turn in the case during convalescence. In the mildest grades one or two vomiting spells that empty the stomach may change the appearance of the individual almost as quickly as did the onset of the unfavorable symptoms. The quick recognition of the condition and the early resort to the stomach-tube treatment saves the majority of these individuals. In my opinion, in these slight cases the intestines may be mainly affected, and in this instance general gaseous distention and obstinate constipation are diagnostic features.

It must be remembered that in many cases the condition of the stomach is merely the result of the condition lower down in the intestine. In such an instance the entire tube of the small intestine and stomach may be involved in the adynamic process. It is probable that in the majority of cases where a large accumulation of fluid in the stomach is met with and the case diagnosed early enough to be relieved and cured, that it is the frequent emptying of the stomach, there thus being nothing to pass down the tube, that makes possible the re-establishment of normal peristalsis in the intestines. If it is true, as Alvarez has pointed out, that it takes a shorter time for food to traverse the intestinal canal when put in above, then the nodal points on the way down take their stimulation from the higher ones. It can then be understood that if a low section of the small intestine is involved in the paralytic process, the constant accumulation of fluids above this point gradually does away with the possibilities of stimuli coming down through the Meissner and Auerbach plexuses, and the more the canal, and finally the stomach, distends, the more paralytic the entire process. In such an instance if the stomach be emptied (that means the emptying of more or less regurgitated matter from the small intestine) there is less bulk and less right angle pressure in the gut below it, and that is the reason why stomach lavage is helpful even though the origin of the paralysis is in the intestinal canal.

In not a few of the cases, particularly in gynecological practice, namely, operations in the pelvis, and also in abdominal operations, the ileus is entirely colonic. There is no doubt that the so-called "gas pains" following operation are partial degrees of paralysis of the colon, although it can be assumed that some of them involve the small intestine as well. Thus, if the stomach is empty, the condition is only a partial status of paralysis so low down in the intestinal canal that the stomach accumulation has not taken place.

Prognosis.—The prognosis in severe cases with typical symptoms is bad, about 70 per cent. of them dying in a short time. It is fortunate, however, that the majority of cases are moderate or that the paralysis is low. The prognosis, however, must be carefully guarded. The cessation of vomiting is not always a favorable sign. This may be due to the patient becoming so weakened and prostrated that vomiting is no longer possible. More reliance should be placed upon the general condition of the patient, and the diminution in the amount and character of the return from the stomach obtained by the stomach-tube. When bile, steapsin and succus entericus and no gastric enzyme are constantly seen, the prognosis continues grave.

When an achylic return slowly develops into one having hydrochloric acid and gastric enzyme, the prognosis becomes more favorable. Should there develop a fecal odor to the gastric contents or a return of fecal substance itself, the diagnosis must be changed to intestinal obstruction below the duodenum.

Fig. 97.—Patient with author's continuous drainage stomach-tube in use.

Treatment.—At once when the diagnosis of post-operative dilatation of the stomach and duodenum or intestine is made, all foods and fluids should be withheld by mouth and starvation with stimulation practised. In the severer cases with a sudden onset, early rectal feedings are not important, the enemata of infusion of coffee or other stimulants being first in order. In every case, both severe and mild, the stomach should immediately be evacuated, preferably by lavage with plain warm water, or a saline solution. My preference is to have this water rather warm—from 110° to 120° F. The washing out of the stomach relieves the pressure excited by the organ and cleans out the fermenting contents and prevents them from further

poisoning the body. This has the advantage of acting as a stimulant to contraction. One must not wait until the symptoms become marked, or temporize with emetics which rarely work in these conditions. The lavage should be frequent, several in twenty-four hours, and in severe cases the distention and accumulation cannot be kept down unless this is done about every three hours. The severer the case, the more frequent the stomach washings, and performing it once in every one or two hours is not too frequent in the beginning. The lavage may have to be kept up for several days, according to the existing conditions. It is safer to lavage too often than not often enough. The author's method of continuous drainage of the stomach is a most useful and comfortable means to drain the stomach, medicate, and later on to feed the patient. Thirst should be combated by proctoclysis or hypodermoclysis. With a favorable turn of the patient rectal feedings may be instituted. As a rule these cases die or get well within a short time, and thus the rectal feedings answer all purposes, and in many instances may be left out altogether for a few hours. The bowels should be moved by enema, preferably a saline solution, and hot turpentine stupes may be used to relieve the distention. No medication of any sort should be given by mouth, not even for the purpose of purgation, as well as for stimulation. All the medication employed should be in hypodermic injection, of which several measures have been used with good results in some cases and not so good in others.

Hormonal is the trade name for the peristalsis hormon discovered by Zueltzer, and is a chemical substance which occurs in all the organs of the body, but especially in the spleen, and which exercises a specific stimulus upon the peristalsis of the intestine. It is administered by deep injection into the gluteal muscles, in an average does of 15 cubic centimeters. Zueltzer, Henle, and Saar have reported observations with this drug even from a single injection. I have used it several times in post-operative paralytic ileus of lesser or moderate degree, usually by intravenous injection of hormonal. In some of the cases the results have been good, but of late some fatal resuls have been reported from its use, and with the change of procedures I have not employed it.

Mention has already been made of the use of salicylate of physostigmine in ½-grain doses. This might be employed in place of hormonal.

Schulz has used atropine successfully in 95 per cent. of 20 chronic cases of paralytic ileus, and in 68 per cent. of 54 cases of mechanical ileus. He claimed for the drug that it regulated the circulation of

the blood through the region and abolishes pain, spasm and vomiting, while it promotes the absorption of the local inflammatory process, and is especially useful in neutralizing the injurious action of the ptomaines which are being produced in the occluded intestine. Atropine is practically harmless, even when the symptoms of atropine intoxication occur. As soon as acute ileus is diagnosed or even surmised, he injects 0.002 gram for an adult and 0.0005 gram for a child, without resorting to preparations of opium. He generally injects, before the atropine, 0.0012 gram of morphine to protect the brain, but does not regard this as indispensable. If the first injection shows no appreciable results or merely improvement of the general condition, without passage of flatus or stools, he repeats the injections systematically, as soon as the patient has recovered sufficiently from the preceding injection. He applies at the same time certain physical measures, oil enemata, hot water bottles, and lavage of the stomach. He advances the very practical idea that the patient should be taken to a hospital, but he advises against operating too early, as the effect of the atropine may be felt even in long neglected cases, while the proportion of recoveries after operative treatment is the same whether the operation is done the third day or in the second week after the first symptoms. Even in desperate conditions he states that the atropine should be injected to improve the heart action and circulation, and to arrest vomiting while stimulating peristalsis. If symptoms of severe intoxication develop, morphine should be given as an antidote to the atropine. My experience has not been as brilliant as Schulz reports, although I have seen it do much good in individual cases. In slight cases it is often successful.

Many cases have been recorded as helped by the use of pituitrin. This seems particularly of value in the case in which the blood pressure has dropped. Bidwell reports the use of pituitrin in laparotomy cases. Beginning 12 hours after the operation he gives ½ cubic centimeter every four hours for three days intramuscularly and from his results he considers it evident that pituitrin has a very marked effect on the muscular coats of the bowels and that it is able to overcome the temporary paralysis due to their exposure at the time of operation. He claims that all patients passed flatus freely within a few hours of the first injection and were free from abdominal pain or distention. It is well when pituitrin is to be used to give it intravenously, preferably in the median-cephalic vein. For this purpose ½ cubic centimeter of pituitrin is slowly injected and when it is going to be efficient, results are noted within a few moments. I have used it a number of times and have found no fatal effects from its use as has

been reported with hormonal. My experience has been that if it does not work, it does no harm.

Taking a case in which *bona fide* mechanical intestinal obstruction and peritonitis have been excluded, one of the very best methods of treatment is trans-intestinal lavage with a hypertonic solution. Recently my associates and myself have employed it with the idea in view to favorably influence the main situation of the adynemia—namely the small intestine. While it was not successful in all instances, in the vast majority within an hour after the solution was run into the small intestine a complete flushing of the bowel had occurred, and in a few hours the patient was entirely over the condition. Altogether 26 cases have been treated, all of them moderate or severe in degree. Of these but three died. Two of these were cases of peritonitis which were not diagnosable excepting at autopsy; one was a case of *bona fide* mechanical obstruction due to long masses of flaccid exudate. It is quite remarkable how quickly the picture of a serious case will change when trans-intestinal lavage has been successful. It is well here that the solution be warmer than usually employed —namely from 110° to 120° F. It is important that before the duodenal tube can be introduced into the duodenum that the stomach be washed clear of its content. By the use of a Jutte tube not much if any peristalsis is required to carry the tip into the duodenum, posture alone being efficient.

Various authors have advised the use of massage and electrical treatments. One, Dieffenbach,[24] suggested the use of the galvanic current from 10 to 15 milliamperes, one pole being in the rectum with the positive pole placed over the right hypogastrium. I have had no experience with depending upon an electrical method of treatment alone, or in fact, any electrical treatment as a part of the other measures employed in a case.

An important item of treatment is the postural method. The half sitting position or lying flat with the head of the bed blocked up has been advised for the type of dilatation which accompanied pneumonia when the cardiac and respiratory symptoms are marked. This posture relieves the pressure in the lower side and in that way relieves the embarrassment on the heart's action and the frequency of respiration. In the post-operative cases it seems logical to suppose that while relieving the pressure on the diaphragm would serve to some purpose in so far as the chest is concerned, and even favor downward drainage from the stomach, at the same time it would also increase the danger of lower position of the small intestine and thus increase the element of mechanical obstruction upon the duodenum. For this reason, eleva-

tion of the foot end of the bed has been advised. Others have favored the side position, usually the right, to encourage drainage of the stomach, and some even—notably Schnitzeler—have employed the prone position with abdomen down.

Summarizing the measures of treatment above mentioned, the following may be stated:

Begin the treatment as quickly as possible by washing out the stomach with a warm solution. If there is much accumulation, wash again in an hour, and effort should then be made to carry out a successful trans-intestinal lavage. In the meantime a dose of physostigmine salicylate may be given, atropine or pituitrin the second time, but these two not employed in the third hour. If the duodenal lavage is not successful, effort should be made to get the bowels to move by medicated enema, in which there is dissolved about 30 grams (1 oz.) of pulverized alum in addition to Epsom salts, oxgall and perhaps turpentine. Of late with me a much more efficacious type of medicated enema is one consisting of equal parts of milk and molasses. The stomach should be washed again, and if it is found that secretions accumulate fast, the continuous drainage method of the author is advised. If the bowels did not move after the cutaneous intravenous administration and the trans-intestinal lavage and medicated enema, one would have to resort entirely to frequent washings of the stomach and withholding foods by mouth and by rectum. After a few hours effort should again be made to get the bowels to move by the above mentioned procedure, and in that way with sustaining measures, such as keeping the patient warm, giving hypodermic injections of strychnine, brandy, etc. a few hours go by. If then there is no bowel clearing within twenty-four to thirty-six hours, the patient should be prepared for operation. On entering the abdomen note should be made as to whether we are really dealing with paralytic ileus, or an intestinal obstruction due to mechanical means, or a peritonitis. The findings would dictate as to the proper surgical procedure. If it is a paralytic case, to me, a drainage tube and insertion into the gut are called for. Then the incision depends somewhat upon the site of the incision of the original operation, under which circumstances the first incision may be used for the entrance into the abdomen. If such is not feasible the choice of the location is in the median line midway between the umbilicus and the symphysis pubis, for in this region a loop of the intestine near the lower end of the jejumum or the beginning of the ileum can be most readily picked up. The operator will select a point in the small intestine approximately midway between the pyloric end of the stomach and the ileocecal

valve. For more on the surgical treatment the reader is referred to works on surgery. But it must be remembered that any surgical method of treatment is attended with grave danger and should be resorted to only after every effort by medicinal means has proven futile. But it is apparently indicated in extreme cases in which death seems impending, as sometimes occurs in paralytic ileus or in ileus due to perforative peritonitis, or in ileus due to diseased conditions of the spinal column, producing a paralyzed state of the intestines.

CHRONIC DIARRHEA.

Diarrhea due to colitis of the catarrhal and ulcerative type, dysentery due to the various forms of dysentery bacillus, the amebic, luetic, cancerous and tubercular forms are treated elsewhere in this volume. In this connection only the gastrogenic, neurogenic, dietetic, simple bacillary, and that due to colonic stasis and glandular disturbance will be considered.

Gastrogenic.—This form of diarrhea is that found in cases of achylia gastrica in which the test-meal shows no free acid, and the total acidity is 10 or under. The condition is considered more extensively in my volume on Diseases of the Stomach. It is a question in my mind whether every true achylia gastrica case is not definitely one of atrophic gastritis. Being careful to eliminate instances in which the psychic effect of test-meal extraction may have had to do with there being no gastric secretion, and those in which there was no gastric secretion for a limited time because of certain constitutional disorders, the study of such cases as may be termed true achylia gastrica correspond so closely to those of atrophic gastritis that I question that a term like achylia gastrica should be continued in the momenclature. There are some neurogenic cases in which an absence of gastric juice secretion persists for a length of time, and while these may be termed achylia gastrica they might better be included in the neurogenic group. It is a mistake in medicine merely to classify cases on the presence of a symptom, because considerations of pathology are liable to be neglected. The old premises of pathologic classification which have stood the test of time are as true today as when first advanced, and this is the safest and best in abdominal work. This point is important because I doubt that diarrhea is an accompaniment of failure of gastric digestion. One of the great factors against it is that only a fourth of the cases of persistent absence of secretion in the stomach have diarrhea as a symptom.

In my opinion achylia is a very common accompaniment to diarrhea, and there is in each instance of the so-called achylia gastrica a direct exciting cause. What may be termed cases of distinct achylia gastrica are those with definite catarrhal colitis, and the absence of gastric juice secretion seems to be a symptom of the trouble in the colon. Here the treatment of colitic condition usually benefits the stomach. Such a colitis is seen most clearly in elderly people, when it is of the atrophic form, and there is liable to be associated with it chronic disease of the pancreas; in which perhaps the interference with pancreatic secretion is a far more important factor in the production of the diarrhea than is the absence of gastric secretion. Carefully studied cases of achylia gastrica discloses such conditions as tuberculosis, atrophic gastritis, chronic disease of the pancreas, malignant disease, parasitic disorders of the bowel, and others. Before a diagnosis of achylia gastrica be made, and it is better that it never should be, a careful search should be instituted as to the cause of the absence of gastric juice secretion. Such causes are usually found. If they are not found, then we may consider that we are dealing with a cause bound up in the mysteries of the nervous system and innervation affecting secretion and motility.

The treatment, therefore, would be the treatment for the cause and since these causes are variable, it need not be entered into here. It may be said, however, that to control the diarrheal symptom in a temporary way massive doses of tannigen, giving 5 grains every hour day and night, for several days, has been beneficial. A starch and vegetable diet, with alkalies and pancreatic extract is often helpful.

Neurogenic.—While neurotic conditions as an explanation of chronic diarrhea are not satisfactory they are seen from time to time. One needs but the experience of examining medical students for degree or for hospital positions to note that the toilet, as well as the urinal, is excessively over-worked by some of the students during the time that the examinations are on. I have had students with whom so many movements of the bowels took place they could not complete their examination on that day or without some medical assistance. Of course, these are individuals of nervous temperament in whom the nervous system becomes easily irritated. In practice, however, it is well not to jump too quickly to neurogenic causes for a chronic diarrhea. For an acute diarrhea perhaps, but for a chronic rarely is it the cause, because it stands to reason that an exaggerated emotional state can not persist long enough to keep up an abnormal peristaltic activity of the colon for weeks and months of a diarrhea. Nothnagel and Peyer[25] report instances of chronic nervous diarrhea.

Some persons will be attacked with gurgling, abdominal pain, tenesmus, and diarrhea as soon as they find they can secure no access to a water-closet, or are locked up in a train for several hours. It should be understood here that such a case is one of psychasthenia, and the treatment belongs to the treatment for that.

Cases have been described of tabes, or intestinal crises, which had diarrhea as a manifestation, or as a reflex from some uterine condition or pregnancy, in which it was assumed by Condio that diarrhea takes the place of vomiting. Nervous diarrhea has also been attributed to excessive smoking, and I have seen several cases of this type. I have also seen it as an hysterical manifestation which existed for some time. One must remember in this connection that there is a very close association between the colon and the nervous system, particularly the emotional sphere of the brain. In a large gastro-intestinal practice one not uncommonly sees a case, usually in a woman of the nervous type, wherein there is a run of several weeks of diarrhea that can only be controlled by rest in bed, and treating the case along neurological lines. It must be assumed in them that this is of the neurogenic type, and may even be classed as chronic. However, even here, careful administration of glandular therapy often brings about most striking results for control of the attacks, and even for their prevention.

Dietetic.—As is well known, certain articles of diet may produce diarrheal evacuation, such as fresh fruit, cucumbers, pickles, cabbage, turnips, beets, etc. In patients of rare susceptibility, milk produces diarrhea in some while in others it constipates. Excess of food or too great ingestion of water or beer with the food may prevent gastric digestion and in that way bring on a diarrhea. An intestinal fermentation or putrefaction acting as an irritant to the mucous membrane of the intestine can likewise cause a diarrhea. The importance of the bacteriology of this will be taken up later on in this chapter. Diarrhea may be associated with constipation, due simply to the bowel content remaining in the canal too long and acting as a local irritant, and in that way bringing on a succession of fluid movements accompanied by colicky pains, bloating, and the development of more or less offensive gases, such as sulphuric and hydrogen.

When one considers the various types, all the way from those who are obstinately constipated, to those whose bowels move too frequently from articles of diet which to the obstinately constipated would cause no symptoms, a very diverse picture, even when no pathology is present, is met with. There are individuals susceptible to certain foods in whom the bowels usually move normally, but if

they partake of these foods too frequent movements of the bowels take place. These food substances act as an irritant to the mucous membrane, producing hyperperistalsis, with an increase in the secretion thrown into the gut. Ofttimes such a patient can tell you what foods produce the looseness of bowels. This is generally accompanied by more or less local disturbance in the abdomen. At times, however, the individual's attention has not been directed to any special types of food—simply the history of diarrhea being present. With such, when the diarrhea attacks have been frequent, it has been a practice with me to test the reactions to food anaphylaxis, using the vaccination method and observing the reactions. Not a few times foods which were least expected to be irritating to the intestinal canal were found to be the offending factors, and which when removed from the diet caused a subsidence of the diarrhea. These vary so greatly that they cannot be classed in any group, but usually they are found in the cereal or vegetable group, the proteins of which would be anaphylactic to that individual. There are many in whom the food factor itself is not as important as the bacteriology of the gut, namely that the food plus the bacteriology is the combined factor producing irritation of the intestinal content. These will be entered into in the next section of this chapter. Of course, when certain articles of food definitely produce a diarrhea they should be eliminated.

Simple Bacillary Diarrhea.—The finding of the gas bacillus (Bacillus aërogenes capsulatus) in excessive numbers in the feces of patients suffering from attacks of acute diarrhea is a fact substantiated at the present time by a considerable number of observations. It is my opinion that in not a few cases of diarrhea, the gas bacillus and the Gram-positive diplococcus, present in large numbers in the intestinal canal, can cause a condition of chronic diarrhea by the production of a hypertrophic colitis with more or less spasm and general irritation in the colon.

This type of diarrhea is best seen in infants, although it is not uncommon in the adult. Usually the history is an acute one, the diarrhea being over in three to seven days. In such an acute case, the onset may be by a chill followed by vomiting and malaise. The temperature may rise for three or four days, a temperature from 100° to 103° being noted, this usually becoming normal on about the fourth or fifth day. Movements are usually frequent and as many as ten or fifteen a day, and with the substance of the diarrhea more or less mucus is seen. Such an attack may be followed by more or less looseness or diarrheal tendency. The streptococcus in the intestine may bring on a diarrhea, and possibly is the causation and contin-

uance of some cases of chronic colitis. Such cases of simple bacillary diarrhea of the acute and chronic types present themselves as a colitis. This catarrhal condition of the intestinal membrane is doubtless a factor in keeping up the diarrhea and could be independent of any special infection or fermentation of the food content which may occur. However, there are instances of infection of the intestinal content in which the intestinal mucous membrane is not enough changed to warrant the diagnosis of a colitis but in which a chronic diarrheal condition exists, the gas bacillus producing a fermentation and the production of butyric and other organic acids being the main factor to be controlled. I feel that this is no longer "merely speculation" as Kendall[26] has suggested, because I have met with it so often and had such striking results when the infection was under control, as to prove it the cause of a chronic diarrhea, even if it is not well known in the medical profession. I doubt that the colon bacillus is ever a factor, either primarily or secondarily. In marked B. coli infections of the gut, as a rule, the mucous membrane is dry and an obstinate constipation exists, instead of it being wet, moist and hypertrophic with a tendency to looseness of the bowels.

In my experience the buttermilk diet as suggested by Kendall is efficacious to a certain extent and perfectly so in the acute case; in the chronic case the best form of diet is one that is high in protein and in which the carbohydrates and fats are in negative amounts. With a high protein diet such as eggs and water, or like the one following, which is a better balanced diet, and in which there is very little or no starch and sugar substances for the gas bacillus to grow and feed upon, much better results are accomplished than with the buttermilk diet:

This diet is a temporary one. Take mostly meats—all forms of beef with the exception of cuts from the shoulder, kidneys, and liver. The same is true of lamb.

These meats should be fresh and taken in a broiled or roasted state. Mutton is permissible but no pork nor veal.

Any kind of fish, broiled, roasted or boiled, may be taken with the exception of shad roe and shell fish.

One may eat eggs in any form.

Any form of cooked, fresh vegetables that are green, such as spinach, beet tops, celery, etc., and any of the salads with French dressing very low in vinegar, may be taken.

Butter and whole milk are allowed, together with any form of simple cheese of the cream variety, such as Philadelphia, Neufchatel, etc.

Eat as much gelatin foods as possible.

Oatmeal and rolled oats are allowed.

One may have breads or crackers made of gluten or rye flour.

Lentils and dried peas are permissible.

There is no objection to an occasional orange, pineapple or strawberries.
The best drink would be chocolate and cocoa.
All kinds of fermented milks may be taken—such as Kumyss, Matzoon, etc.

Medically, the condition requires alkalies, either by the administration of bicarbonate of soda or bismuth subcarbonate by mouth, or by bicarbonate of soda enemata, using about two tablespoonfuls of bicarbonate of soda to two quarts of warm water. The use of peroxide and water enemata (1 to 2 ounces to 2 quarts), or an enema of permanganate of soda solution, answers a good purpose here.

Colonic Stasis.—A definite group or groups of cases complaining of diarrhea may, with modern methods of diagnosis, especially the Roentgen rays and sigmoidoscope, be found to have an underlying colonic stasis. What is interesting in these individuals is that although they will have apparently normal movements, a considerable portion of the stool will consist of small balls (spastic feces), and an examination of the sigmoid in spite of several previous movements during the morning shows a considerable filling of well formed feces in the form of these balls. Some of these cases are attended by an abnormal tightening of the sphincter muscle, with or without irritable fissures or hemorrhoids. Others again will show a distinct fecal impaction, and fluoroscopic or Roentgenographic examination shows a dilated colon with a passage through a portion of the impacted area. This type of stasis constipation is seen time and time again, particularly in corpulent people. We have also a type of diarrhea in people with a dilated cecum due to long-standing chronic intestinal putrefaction. Such a cecum may have a stasis, retaining its content an abnormal time, permitting fermentative processes to be set up, giving rise to intermittent or persistent diarrhea.

All of such cases are treated best by considering them as constipated and being given an anti-constipation diet in which the amount of cellulose and roughage is high, and bran, agar, prunes, and other food substances are taken at regular intervals during the day to accomplish this. Of course, where there is a chronic excessive intestinal putrefaction or fermentation the case would require individual treatment both as to diet and vaccine. This depends upon the bacteriological findings of the stool examination.

Glandular Disturbance.—Not a few people who have chronic diarrhea have distinct evidences of hyperthyroidism, even definite exophthalmic goiter. We deal here simply with a hyperperistalsis without failure of gastric or intestinal digestion, without colitis and without stasis, Many cases of diarrhea in a neurotic subject have their origin in a disturbance of the thyroid function. Careful search

should be made for the characteristic signs associated with the hyperfunction of this gland. Vagatonia comes in here to account for some of the cases.

Chronic Pancreatitis.—Any condition which causes a diminution or an absence of secretion from the pancreas is liable to be attended by large, soft, fatty stools, and may be not always associated with diarrhea. Sometimes a glycosuria with a high degree of intestinal putrefaction and colitis are present with deficient pancreatic secretion.

There is some question as to whether a distinct interference with the secretion of the pancreas can cause a diarrhea. My belief is that it can in a few instances, because I have given pancreatic substance, usually in the form of food, uncooked, and found that the diarrhea was perfectly controllable.

Roberts,[27] in a clinical study of chronic diarrhea believed that five of his cases were due to an incipient form of Addison's disease characterized by a marked secondary anemia with high leucocyte count, disproportionate loss of flesh and strength, low blood pressure, and a slight general bronzing of the skin without definite changes in the mucous membrane. In three of his cases excellent results were secured by the use over a long period of nucleoprotein of the adrenal, although other forms of that gland failed to have any effect upon the diarrhea.

ENTEROGENIC CYANOSIS.

This was first described by Stokvis[28] in a patient who suffered from severe enteritis with pronounced cyanosis of the skin and visible mucous membranes, together with a swelling of the terminal phalanges. Spectroscopic examination of the blood shows a black band in the red, the blood containing either methemoglobin or sulphemoglobin. These were due to poisonous substances formed in the intestine which transformed part of the hemoglobin into methemoglobin. Talma[29] and Van der Bergh[30] have reported cases, the latter being due to sulphemoglobin from sulphid hydrogen in the blood absorbed from the intestine.

Several cases of sulphemoglobinemia have been associated with obstinate constipation, the relief of which has been followed by improvement.

It is a well-known fact that methemoglobin results from drug poisoning, such as large quantities of bismuth subnitrate, or from intoxication due to absorption of nitrites from the intestine in chronic diarrhea. Sulphemoglobin seems to be associated with chronic constipation, the results of hyperformation or hyperabsorption of sul-

phurated hydrogen, or of the presence of an abnormal reducing agent in the blood, acting with a small trace of sulphurated hydrogen. Stifel has recently reported a case due to nitro-benzine poisoning from shoe dye.

For the diagnosis of this condition a spectroscopic examination of the blood is necessary. Without this it might be suggested by the presence of abdominal symptoms plus a marked status of cyanosis of the skin and mucous membrane.

Very little is known about the condition. The wisest method of treatment is prompt purgation, withholding foods as long as possible, and then to restrict the proteins, using milk, purgatives, enteroclysis, and the general treatment for sustaining vitality.

ENTERALGIA
(Intestinal Colic).

In a way it is an unfortunate thing that the term enteralgia has ever been used in connection with medicine. It merely means a pain that may originate from inflammation of the intestinal wall or the peritoneal coat, or a colic. It is only a symptom, and should never be used as a diagnosis in any way.

It is caused by any of the organic lesions of the intestine, excess in or improper articles of food, cold drinks, substances causing marked gas formations, fecal accumulation, intestinal worms, foreign bodies, gall-stones, large quantities of mucus as in mucus colic, colds, gout, exposure to wet, ulcers, internal strangulation of the bowels, stenosis, purgatives, lead and copper poisoning.

It therefore must be plain that the diagnosis should be that of the causative condition of the enteralgia, and the treatment likewise is based on the cause.

The author thinks it is unwise to enter into a discussion of the treatment of enteralgia because such suggestions as might be made may be applied to a case of acute appendicitis, volvulus or some other serious condition.

INTESTINAL SAND.

The first case of intestinal sand was reported by Laboulbene[31] in 1873. He described a sandy substance in the feces resembling brown or yellow sand, which he believed was of vegetable origin. Since that time numerous cases have been reported which had more or less association with clinical manifestations, and attempts have been made to show that the sand was a part of various forms of diathesis. Re-

cently Myer and Cook[32] studied the literature, and came to the conclusion that intestinal sand was not a part of any clinical entity but that, in their own case, it resulted from bananas. They found that after eating bananas, sand invariably appeared in the feces after twenty-four hours, and often it continued for several days. I have seen one case wherein three or four bananas would cause an output of intestinal sand amounting to two or three tablespoonfuls. The grains are often found in chains. These chains correspond in arrange-

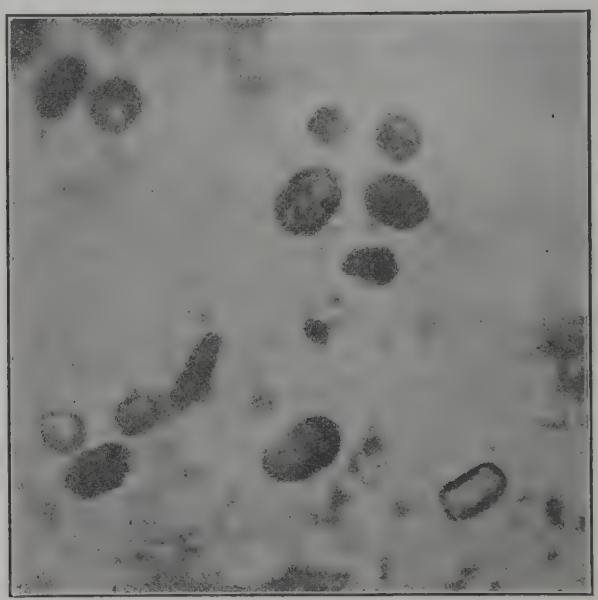


Fig. 98.—Intestinal sand in feces. ×62½. (*Talbot.*)

ment and shape to the cylindrical cells in the milk-ducts of the banana. These cells contained highly refractive resin balls or masses, which were suspended in a fluid rich in tannin.[33] Myer and Cook believed that these resin balls were affected by the secretion of the stomach and intestine in such a way that the resin was hardened and an insoluble tannate formed. They were unable, however, to reproduce the sand by artificial digestion or by placing the banana in fecal material.

One not uncommonly meets with sand in stools of patients. Usually the quantity is not very great and escapes the observation of the patient. In most of these individuals, however, the presence of the so-called intestinal sand is not due to the eating of bananas.

Symptoms.—Attention is drawn to the condition by black or round sand-like particles intermittently mixed with the fecal material, so that the stool may look peppered. Under the microscope they show a small amount of fat in the form of soap, generally a few meat fibers, the sand is somewhat heavier than the fecal material, and it may be obtained in a pure condition by frequent washing and straining through a sieve and decantation. It is impossible to separate the sand entirely from the cellulose remains. It has a brown sandy appearance and feels gritty between the fingers.

Talbot[34] examined sand under the microscope and found one kind to consist of a colorless variety which resembled calcium or magnesium phosphate, and another irregular or oval, sometimes crystalline with rough edges and sometimes smooth. The color varied from a light yellow to a beautiful deep claret red. The crystals were translucent but not transparent. The grains were hard and brittle, and when crushed under the cover-glass, still retained their crystalline appearance. There is no doubt that the composition of this sand varies in different cases.

Marquery[35] found organic matter 72 per cent., inorganic 28 per cent. Dieulafoy[36] found water, 11.25 per cent.; fecal material, 22.6 per cent.; phosphoric acid, 17.56 per cent.; calcium 26.22 per cent.; magnesium, 14.05 per cent.; silica, 5.68 per cent. Myer and Cook found moisture 17 per cent.; organic matter, 95.8 per cent.; nitrogen (Kjeldall), 2.5 per cent. The inorganic portion consisted mainly of the phosphate of calcium. The sand in Talbot's case showed the following analysis:

Nitrogen	3.0 per cent.
Total fat	5.0 per cent.
Ash	17.4 per cent.
Calcium	7.4 per cent.
Magnesium	2.0 per cent.
Phosphates	present.
Material soluble in alcohol after hydrolysis (resinous)	14.0 per cent.

The patient, who may be quite young, usually complains of digestive disturbances during which there is a loss of appetite, nausea and sometimes vomiting. A child may become cross, irritable and have a slight fever. The attacks are usually frequent, coming on in spells, varying from one week to three or four months.

Treatment.—In my opinion this so-called intestinal sand represents a status of intestinal toxemia, usually with a low degree of enteritis, and sometimes entero-colitis. The treatment for the condition is that for the underlying cause, whatever its form.

During an attack an enema with a dose of castor oil usually brings about a quick recovery from the symptoms. The withholding of banana as a food is not essential because in most of the cases in which intestinal sand occurred bananas had not been eaten.

LEAD COLIC.

Among workers in white lead factories, plumbers, painters and glaziers this disease is common. Accidental poisoning may occur, mainly by drinking water which has passed through lead pipes or been stored in lead-lined cisterns. Wines and cider which contain acids quickly become contaminated in contact with lead. All ages are attacked but children are relatively less liable. Usually the condition occurs in the adult between 30 and 40, women being more susceptible than men. With women the disease is liable to cause miscarriage, and it is rare for a woman working in lead to carry a child to time. It also destroys the reproductive powers of men. It can bring about death by bringing on encephalopathy, Bright's disease, cerebral hemorrhage, paralysis, phthisis, and various maladies, such as pneumonia, heart disease, aneurisms, etc.

In chronic lead poisoning the metal is found in various organs; the muscles are yellow, fatty and fibroid. The nerves present the features of a peripheral degenerative neuritis. In the acute fatal cases there may be the most intense enterocolitis.

Symptoms.—The symptoms of the acute form need not concern us here. In the chronic type there is a moderate grade of anemia, with the constant presence of nucleated red blood-corpuscles even when the anemia is of very slight grade. A blue line on the gums (which in my experience is more definitely a bluish-gray line) is a valuable indication, but not invariably present. The color is due to a precipitation of lead in the form of lead sulphide by the action of sulphureted hydrogen from the tartar of the teeth. The same may be seen about the anus due to the effect of sulphureted hydrogen gas in the intestine causing a sulphide precipitation.

The most common of all symptoms of lead poisoning is colic, which is often preceded by gastric or intestinal symptoms, particularly constipation of a severe degree. The pain is usually general in the abdomen, the colic paroxysmal, and relieved by pressure. There may be vomiting, and during the attack, as Riegel noted, the pulse is increased in tension and the heart's action retarded. Attacks of pain with acute diarrhea may recur for weeks or even for three or four years. Certain cases with colic may present the features of an acute

intra-abdominal inflammatory condition and mistakes have often been made supposing that an appendicitis or intestinal obstruction existed. In this connection it may be recalled that chronic lead poisoning should not be diagnosticated on the presence of generalized abdominal tenderness and attacks of colic alone.

Lead palsy is present in about half of the cases, the upper limbs most frequently affected. However there may be a slow chronic

Fig. 99.—Bluish gray line at teeth in chronic plumbism. Best noted in upper gingival margin.

paralysis gradually involving the extremities, beginning with the classical picture of wrist-drop. The electrical reactions are those of lesions of the lower motor segment, and reaction of degeneration in its different grades may be present, depending upon the severity of the disease.

The cerebral symptoms are numerous. These are symptoms of delusion and maniacal excitement, a transient delirium, attacks of unconsciousness, and perhaps convulsions. Optic neuritis or neuro-retinitis may occur. Hysterical symptoms occasionally occur in girls. Convulsions are not uncommon; arteriosclerosis with contracted kidneys and hypertrophy of the heart are common.

Treatment.—Prophylactic measures should be taken at all lead-works, but as a rule, employees are not careful. Cleanliness of the hands and of the finger-nails, frequent bathing, and the use of perfect measures of ventilation are in order. In the instance of lead colic, local applications of heat may control the abdominal pain, but morphine may be required. Difficulty is usually met with in getting the bowels to move, and often quite drastic measures, together with medicated enemata, are necessary. Iodide of potassium 5 to 10 grains (0.3 to 0.6 gm.) three times a day are in order, and an occasional morning purge of magnesium sulphate should be given. For the anemia iron in inorganic form is best.

TYPHOID AND PARATYPHOID FEVERS.

It is not the purpose of this volume to go into a complete description of typhoid fever, which is a systemic disease, but simply to mention the abdominal symptoms ordinarily met with.

Symptoms.—The abdominal symptoms of typhoid fever usually begin about the second week, but may not be present during the entire course of the illness. When present, they consist of diarrhea, tympanitis and tenderness, the tenderness mostly being located in the region of the ileocecal valve. When they exist in the second week, they usually are aggravated in the third, at which time perforation and hemorrhage may occur. Along about the fourth week, the diarrhea stops, the tongue cleans, and the desire for food returns. In marked cases the fourth and even the fifth week presents an aggravated picture of the third, in which instance the abdomen usually becomes more distended. Some cases begin with incessant vomiting and pain, leading to the suspicion of poisoning, or perhaps there is the suggestion of the presence of appendicitis, but nausea and vomiting are not common. Diarrhea is present in 20 to 30 per cent., and is due less to the ulcers than to the associated catarrh. Hemorrhage of the bowels is a serious complication, occurring in about 7 per cent. of all cases. Meteorism is a frequent symptom, not serious if of moderate grade. Sepsis is usually of ill omen. Abdominal pain and tenderness are present in three-fifths of the cases, and pain alone in only about one-third.

Perforation in typhoid is not uncommon. It is usually indicated by a sudden sharp pain of increasing tendency, often paroxysmal in character, with tenderness on pressure and perhaps muscle rigidity. A friction sound may be present within a few hours of the onset of the perforation. The picture in the end is that of a general peritonitis,

which, however, may come on without perforation of the bowels due to extension from an ulcer or by the rupture of a softened mesentery gland.

PNEUMONIA AND PNEUMOCOCCIC INFECTIONS.

In these gastro-intestinal symptoms may be present, such as the tongue white and furred, and in severe cases when the general condition is reduced, it rapidly becomes dry (typhoid tongue). Vomiting is not uncommon at the onset, particularly in children; anorexia is the rule; constipation is more common than diarrhea. A dangerous symptom is meteorism, which is probably due to a myasthenia of the intestine from the toxic poisons affecting the nerves in the muscle sheath. When present it indicates a marked general toxemia, and when present calls for purgation and most active elimination in every way. The liver may be depressed or enlarged from an engorged right heart or as the result of the infection.

ADDISON'S DISEASE.

This disease is due to a fibrocaceous lesion, tubercular in character, involving the suprarenal, simple atrophy, interstitial inflammation of the suprarenal leading to atrophy, or malignant disease. Cases of blood extravasation into the suprarenal bodies have been reported. As is well known, it is a disease characterized by muscular and vascular asthenia, irritability of the stomach, pigmentation of the skin, and in well-developed cases is invariably fatal.

The condition may set in with attacks of nausea and vomiting, usually spontaneous in character, a cause not being discoverable. More or less anorexia is usually present, and in the end there may be pain in the abdomen with retraction, and even features suggestive of peritonitis. The gastric symptoms, as a rule are variable throughout the course, and when present are the colloquial ones of digestive disturbance. The gastric symptoms, however, may be absent, although attacks of diarrhea are frequent, usually coming on without obvious cause. The diagnosis is usually suggested by the presence of the pigmentation of the skin and mucous membrane, and perhaps the progressive asthenia.

LOCOMOTOR ATAXIA.

This condition is characterized by incoördination of locomotion, sensory disturbances, trophic changes and involvement of the special senses, particularly the eye. As is well known, it is a wide-spread

disease, more frequent in cities than in the country, and is due to a posterior spinal sclerosis. In all probability it is due to syphilis.

It is not the purpose here to enter into a lengthy description of locomotor ataxia, but simply to present the abdominal disturbances not infrequently met with and often mistaken in diagnosis. The most remarkable sensory disturbance in the tabetic are the crises, which are severe paroxysms of pain referred to various viscera in which the most common are the gastric and laryngeal. The gastric crises may occur early and persist as the most prominent feature. Usually they are sudden in onset, with a pain of a severe burning and twisting type in the epigastrium, radiating to the back and behind the sternum. In the presence of a seizure, vomiting shortly follows the pain, this being independent of food. The attack is usually accompanied by more or less of a collapse and shock, pallid skin, sweating, cold extremities and a small pulse. It being best described as an attack of abdominal angina pectoris.

In any case presenting itself with abdominal symptoms in which there is a long history or one of some weeks or months during which these may be described as having been acute, care should always be taken, even in women, to note if the reflexes are present, whether optic atrophy exists, the Argyll-Robertson pupil, the Rhomberg symptom, atrophic changes in the skin, and perhaps evidence of an ataxia.

INTESTINAL HEMORRHAGE.

The significance of intestinal hemorrhage is in connection with the condition producing it, although the hemorrhage may be so alarming that definite means for its contol is important irrespective to the cause. Among the conditions in which intestinal hemorrhage is noted are dysentery, typhoid, yellow fever, malarial poisoning, ulceration of the intestine (usually the colon from whatever cause), cancer, scurvy, purpura, traumatism, volvulus, intussusception, hemorrhoids, excessive use of laxatives, venous hyperemia due to diseases of the heart, lungs or liver, isolated venous varicosities, arterial aneurysms of the intestinal wall, aneurysms in adjacent arteries (such as of the hepatic, from which blood may enter the bile-passages and be passed by the bowel) and after operations. It is met with also in some cases of pernicious anemia, leukemia, septicemia, icterus, phosphorus poisoning and erysipelas.

There may be symptoms of collapse with those of internal hemorrhage, no blood being passed from the rectum for some hours. Usually, however, when the quantity of blood in the bowel is large, it

acts as an enema or as a laxative and soon makes its appearance. Hemorrhage, on the other hand, may be minute, such as is met with in cases of ulcer of the stomach, duodenum, or carcinoma in the upper levels of the gastro-enteric tract. Here, of course, the blood would have to be tested for by chemical means.

Postoperative gastro-intestinal hemorrhage may be encountered after any surgical interference, no matter of what nature, but unquestionably it is more prone to occur after an abdominal operation, particularly when the interference has been for the relief of some lesion of the digestive tract. This means that it must be due on the one hand to local factors, such as primary lesions of the digestive tract or peritoneum, or on the other hand to general causes such as infection, intoxication, or perchance to reflex disturbances.

Infection, be it localized or general, is the most common etiological factor, although it may occasionally be difficult to assign the respective parts played by septicemia and chloroform poisoning when the latter drug has been used as the anesthetic. In point of fact chloroform may not only set up hemorrhages in a predisposed soil, but it is likewise quite capable of determining by itself a sanguineous dyscrasia with a gastro-intestinal localization.

The other major etiological factors are traumatic or mechanical lesions of the digestive tract, which may or may not be attributable to a defective technique, such, for example, as prolonged operative maneuvers on the omentum and extensive resections of this viscus. The nature and seat of the lesions of the digestive tract are very variable, according to the pathogenesis of the hemorrhage. The most frequent, however, is a hemorrhagic erosion or deep ulceration almost always seated in the gastric or duodenal mucosa. In 25 per cent. of autopsies no gastro-intestinal lesion could be discovered.

The clinical picture of these hemorrhages also varies greatly, and the most sagacious medical mind will be quite unable to make a diagnosis of the exact mechanism of the hemorrhagic complication unless all details of the operative interference and symptoms offered by the patient are passed in review, not overlooking the time of the appearance of the loss of blood.

The prognosis of postoperative gastrointestinal hemorrhage is subordinated to both the amount of blood lost and the gravity of the concomitant symptomatic ensemble. Generally speaking, a very reserved prognosis should be made, because out of a total of 249 cases reported by Fourdinier, death occurred in 130; in other words, a mortality of 52 per cent. Purves found a mortality of 72.5 per cent. while Busse came to a mean death rate of 55 per cent. These hemor-

rhages especially demand a reserved prognosis when they arise after operations not involving the digestive tract, and out of a total of 15 cases of extra-abdominal operations where this complication occurred 14 died. In purely abdominal operations the prognosis is less gloomy, as out of a total of 74 cases there were 44 deaths, or a mortality of 59 per cent. In operative work on the digestive tract, the prognosis, although in itself serious, may be considered as relatively benign, because out of a total of 160 gastro-intestinal operations there were 72 deaths, or 45 per cent. Simple hernia operations followed by gastro-intestinal hemorrhage have given a mean death rate of 40 per cent. while in the case of strangulated hernia it is found to be 37 per cent., and 50 per cent. in appendicitis. Therefore, according to Sauvé, one should separately consider early hemorrhage arising during the first four days following the operation, and late hemorrhage occurring after the fifth day. In Busse's statistics there were only 30 deaths out of a total of 56 cases of early hemorrhage, while for late hemorrhage the mortality reached nearly 80 per cent.

Treatment.—In the presence of hemorrhage absolute rest is essential, and this rest should be so complete that the patient should not be permitted to raise his arm or be moved about in the bed, such as for the use of the bed-pan, etc. No foods nor fluids should be allowed by mouth and the room should be kept quiet. The best internal hemostatic we possess is morphia given in the form of subcutaneous injection, and the dose should be a full one according to the age of the individual. When there is collapse, stimulants should be given, and if necessary a hypodermic injection of camphor in oil. Where the circulation has become depleted, the general condition of the patient manifesting this distinctly, an injection of salt solution beneath the skin or directly into a vein may revive the failing heart, but should be done only in case of emergency. The injection of blood serum, usually horse serum, is sometimes very efficacious in controlling hemorrhage. Various proprietary drugs are used for the purpose of controlling hemorrhage, but after a generous experience I question the efficacy of any of them. A rectal injection of a 5 per cent. solution of gelatin, one liter being injected at the temperature of 122° F. in four portions is often of value. Usually it is wise to elevate the foot of the bed during the first eight to twenty-four hours and foods by mouth should not be given until it is apparent that the hemorrhage has ceased.

Surgical treatment is occasionally indicated, but usually the care of the case remains strictly medical. In certain circumstances, prophylactic measures should be carried out in order to forestall the possible occurrence of a post-operative gastro-intestinal loss of blood.

References.

[1] Braun and Boruttau: Deutsch. Ztschr. f. Chir., 1908, xcvi, S. 544.

[2] Von Khantz: Arch. f. klin. Chir., 1909, xxxiii, S. 448.

[3] Clairmont and Ranzi: Arch. f. klin. Chir., 1904, Band lxxiii, S. 696.

[4] Kukula: Ibid., Band lxiii, S. 773.

[5] Roger and Roger: La Revue Scientifique, January 19, 1907, vol. i; Presse méd., January 4, 1911.

[6] Roger and Garnier: Archiv. de med. Experiment, July, 1906; Rev. de Med. Aug. 10, 1906; Presse méd., May 23, 1906.

[7] Murphy and Vincent: Boston Med. and Surg. Jour., Nov. 2, 1911.

[8] Guibe: Presse méd., April 3, 1909.

[9] Braun and Boruttau: *Loc. cit.*

[10] Korentchevsky: Russk. Vrach, 1908, p. 1572 (Cited by Guibe).

[11] Clairmont and Ranzi: *Loc. cit.*

[12] Braun and Boruttau: *Loc. cit.*

[13] Roger and Garnier: *Loc. cit.*

[14] McClure.

[15] Vidal: Rev. de Chir., Oct. 10, 1900, xxii; Congres. Français de Chir., 1905, xxviii, 1237.

[16] Baracz: Archiv. f. klin. Chir., Band lviii.

[17] Draper: Jour. Am. Med. Assn., Oct. 21, 1911; Johns Hopkins Hosp. Bull., 1909, p. 838; Amer. Jour. Med. Sc., vol. cxxxvii, p. 725, and Jour. Am. Med. Assn., 1910, liv. 5.

[18] Whipple, J. H.: "Intestinal Obstruction a Proteose Intoxication," Jour. Am. Med. Assn., Aug. 7, 1915, p. 476.

[19] Dragstedt, L. R., Moorhead, J. J. and Burcky, F. W.: "The Nature of the Toxemia of Intestinal Obstruction," Proc. Soc. Exper. Biol. and Med., 1916, 14, 17.

[20] Jackson: Surg. Gynec. and Obstet., Sept., 1909, p. 278.

[21] Cheever, David: Jour. Am. Med. Assn., July 26, 1913.

[22] Deaver, John B.: New York State Jour. of Med., Sept. 1908, Vol. 8, No. 9.

[23] Cannon and Murphy: Jour. Am. Med. Assn., Sept. 7, 1907, pp. 840-843.

[24] Dieffenbach, William H.: Jour. Am. Med. Assn., April 1, 1911.

[25] Nothnagel and Peyer: Wiener Klinik, 1893, Heft. 1.

[26] Kendall: Boston Med. and Surg. Jour., Jan. 18, 1912, vol. clxvi. No. 3.

[27] Roberts: Read at the Annual Meeting of the American Gastro-enterological Association, Baltimore, Md., May 10, 1915.

[28] Stokvis: Festsch. f. v. Leuden, 1, p. 597, 1902.

[29] Talma: Tijdschrift voor Geneesk., li, p. 721, 1902.

[30] Van der Bergh: Deutsch. Archiv. f. klin. Med., xciii, p. 86, 1905; also Berlin. klin. Wochenschr., No. 1, p. 7, 1906.

[31] Laboulbene: Bull. de l'Acad. de med., November, 1873.

[32] Myer and Cook: "Intestinal Sand: the Banana one of its Sources," Am. Jour. Med. Sc., 1909, cxxxvii, 383.

[33] Bary: Vergleichende Anatomie bei Phanerogamen und Farne, 1877, pp. 153, 160, 451, quoted by Myer and Cook.

[34] Talbot, Fritz B.: Jour. Am. Med. Assn., July 26, 1913.

[35] Marquery: Assoc. française pour l'avangement des sciences, 1879, p. 878.

[36] Dieulafoy: Bull, de l'acad. de med., Paris, 1879.

CHAPTER XVI.

Constipation.

CONSTIPATION, like edema or cough, is simply an irregularity of function, and therefore a symptom. As a symptom it is the commonest disorder of the human being, and much has been written upon the subject even in very ancient times. Various opinions as to its importance are held by different observers, some believing that it is the commonest cause of serious conditions possible to arise in the body, others that its importance has been overestimated.

To the lay mind constipation is the condition of absence of daily movements of the bowels. With people who are habitually correct in this way there is a passage of feces regularly at about the same time each day, the stool in consistency formed in its first part and soft or mushy at the end. It may be taken as a rule that a person who has regular movements of the bowels at the same time each day, the latter part of the stool being mushy or semi-solid, that this individual is not constipated, this also holding true in those who have more than one stool a day. An important thing also is that where no constipation exists there is a feeling of complete satisfaction and emptying of the rectum after each evacuation. In this connection, however, it may be recalled that regular movements of the bowels may exist in which only the lower end of a packed left colon is delivered, there remaining in the sigmoid flexure and descending colon perhaps a much larger quantity than has been passed. In such instances it must be concluded that constipation exists, and that merely the lower end of the columnar mass had been pushed through because of the pressure of a large amount of feces accumulated above.

The causes of chronic types of constipation are numerous, and in each instance all possible factors must be carefully investigated, and if possible, corrected.

Habits.—There is no doubt about it that ignorance, laziness and false modesty are largely responsible for constipation. By ignorance I mean that state or absence of state of mind, found most often in women, particularly in the younger ones, in which the necessity or importance of regular movements of the bowels has never been inculcated, and if so, was never believed. It is a well known fact in rectal work that common is the male in whom no feces is present in

the rectum, but such a condition is almost never met with in the female. Here, feces in more or less amounts is invariably encountered, suggesting most plausibly that women do not attach importance to regular movements of the bowels as do men. Then women have a fashion or habit of not talking about themselves in these ways, and are rarely very observing as to whether the stool passed seems to be a complete evacuation or whether that sense of being empty after the stool exists.

By laziness I mean a carelessness in responding to the call of Nature, and holding back waiting for a more favorable time and place, which if continued long enough will eventually increase the constipation rather than be helpful to it. There usually is a complete lack in these individuals of any effort to establish a regularity of movement of the bowels, and commonly the slightest reason is taken advantage of for deferring this act. When one considers that this is more often the case among the wealthy than among the working classes, people with whom the mode of living and a sedentary life have a bearing, together with worry and mental strain having an influence, the importance of lazy habits is manifest. In this same connection as a cause of chronic constipation may be mentioned false modesty, although it has been my observation that of late years women and men are getting away from this, usually having the faculty of under some subterfuge departing from the company of others long enough to repair to the toilet.

It was my belief at one time that the people who lived in cities, because of the more or less artificial existence, were more commonly constipated than those who lived in country districts. But in my travels about and checking up the subject with populations here and there, I have come to the conclusion that there is very little difference. Evidently a city person can by regulation so habituate himself that he or she can attend to these calls of nature in a satisfactory and timely way, and that such constipation as may come from the absence of sufficient fresh air and exercise is obviated by the more ample diet which the city person can enjoy over his country brother and sister.

Diet.—There is no doubt that faulty diet is largely responsible for habitual constipation, or, I might rather say for the regulation of the bowels in one who because of sedentary life and otherwise cannot live normally. The factors which have to do in bringing this about are many. Some people do not eat enough for bulk in the intestines to stimulate sufficient peristalsis for evacuation. Others eat too much of the kinds of food in which the native proteins predominate, which

have a low stimulation power on the peristalsis. There is no doubt about it that bulk of food is necessary for what might be termed regular daily movements. The use of cane sugar in large quantities is distinctly a constipating factor, and the same may be said of carbohydrates, and these largely predominate in many diets. Very often individuals are met with who do not take sufficient fluid in the course of the day. Water is one of the best intestinal stimulants we have, and while excessive drinking is wrong, too little drinking is also wrong, because then the emunctories of the body have difficulty in carrying on their normal functions.

Another important factor in the dietetic production of constipation is eating at irregular times. If one keeps in mind the mass movements of the colon stimulated by the taking of food, the importance of stated hours for the taking of meals is brought forward. The most delicious articles of food are usually the most constipating and many people do not take sufficient of fruit juices or roughage, for such foods as are high in cellulose accomplish an increase in bulk and a stimulation of the normal peristalsis.

ATONY OF THE COLON: DRY COLITIS.
(Deficient Function.)

In these conditions constipation is commonly present. The atonic abdomen with perhaps a prolapsed organ in which more or less atony of the right colon or deficiency of haustral contraction in the left exists, comprise a large factor in the constipation cases. To diagnose such, careful Roentgenographic observation of the colon must be made as well as direct observation of the mucous membrane of the rectal dome and perhaps the sigmoid. These conditions are usually found in the indolic and mixed types of chronic excessive intestinal putrefaction and in these the constipation is merely symptomatic. It may be said, however, that dry colitis involving the left half of the colon, the sigmoid and the rectum is commonly secondary to a long-standing chronic constipation, because I have observed that when it is possible dietetically and by changing the routine of life to correct the constipation, in the course of three or four months the mucous membrane takes on a healthy appearance.

SPASTIC CONSTIPATION.
(Hyper-function.)

There are many cases of constipation due to irregular spasm usually found in the left half of the colon. Roentgenographic obser-

vations by means of a clysma are necessary to diagnose these. They are usually present in cases of saccharo-butyric putrefaction, and the constipation there is due to irregular haustral contractions which are so hysterical that they anchor the mass rather than propel it forward in a normal way.

FLEXURE DEFECTS.

(Mal-position.)

One not uncommonly sees marked constipation in cases which have a falling forward of the hepatic flexure, sometimes the flexure being so low that it almost occupies the ileocecal region, the ascending colon doubling on itself. Marked constipation may also exist where there is sharp angulation at the splenic flexure, or where the flexure at the ileo-pectineal line is sharply bound down by adhesions or a very short mesentery. These angulation cases are further found in marked prolapse of the transverse colon, particularly in those in which the transverse colon ascribes a V shape in the abdomen. In this connection the importance of loops at the splenic flexure or in the sigmoid are important as causes of constipation.

SENILE CONSTIPATION.

(Exhausted Function.)

With people who are advanced in years constipation is a common factor. Such individuals no longer robust, and deficient in stock of vitality, generally living sedentary lives, have a deficiency in not only the motor power of the hollow viscera of the abdomen but the secretion as well. Careful observation in such individuals of the presence of a change in the bacteriology in the gut, which is no doubt a factor in the production of constipation with them, should be made.

STRICTURES.

Strictures, benign and malignant, providing the latter have not become ulcerated, are common causes of chronic constipation. Such strictures may be due to chronic invagination, tumors without or within the gut wall, strictures caused by bands in the peritoneum binding down the gut or perhaps bridging across, somewhat occluding its lumen.

Diseases and conditions of the stomach, such as hyperchlorhydria, ulcer, cancer, dilatation and achylia gastrica, may cause chronic con-

stipation. The same is true in obstruction to the entrance of bile into the intestine or deficiency of bile, diseases of the heart, lungs, liver and kidneys which bring on an intestinal hyperemia and congestion of the portal system which may retard the peristalsis. In quite a few chronic diseases of the pancreas constipation is met with, the same being true in diabetes, anemia and sclerosis, and also in many diseases of the brain, the spinal cord and of the nervous system. Acute febrile conditions are usually accompanied by constipation.

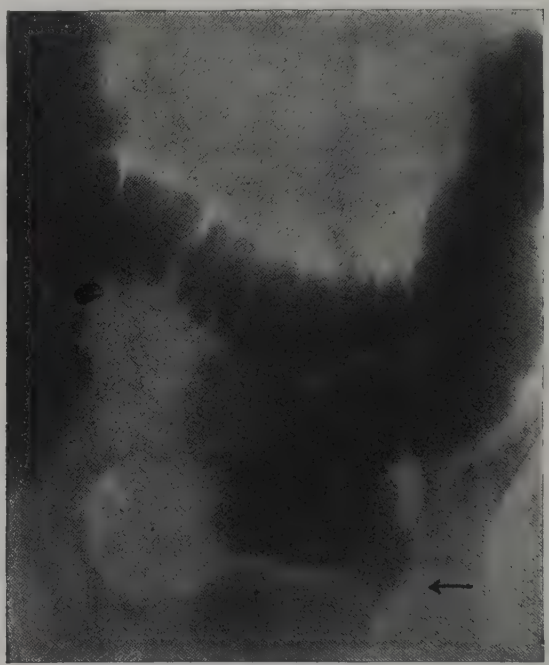


Fig. 100.—Adhesions of pelvic colon at pelvic loop on left side near pelvirectal junction. (*Case.*)

What is important in connection with the subject of constipation is the many cases which are due to rectal causes. It is unfortunate that these are not often enough recognized by medical attendants, because many of them are most amenable to treatment. Among such may be mentioned the following:

HYPERTROPHIED O'BIERNE'S SPHINCTER.

Constipation frequently arises from irritation and thickening of this band of muscular fibers which is located at the rectosigmoidal

junction. Here one meets with feces which accumulate in the sigmoid flexure which are prevented from reaching the rectum owing to the frequent and persistent contraction of the sphincter as soon as the stimulus reaches it.

Fig. 101.—Pelvic colon adhesions with interference with colon evacuation. (*Case.*)

HYPERTROPHIED RECTAL (HOUSTON'S) VALVES.

The rectal valves may become hypertrophied as the result of chronic colitis, ulceration, and other affections of the lower bowel, and when they do they render rigid the lumen of the gut and prevent the feces from reaching the anal outlet. Under such circumstances, lumps of fecal matter can often be seen or felt resting on the upper or concave surface of the valves.

Fig. 102.—Rectal constipation due to inflamed hemorrhoids. (*Case.*)

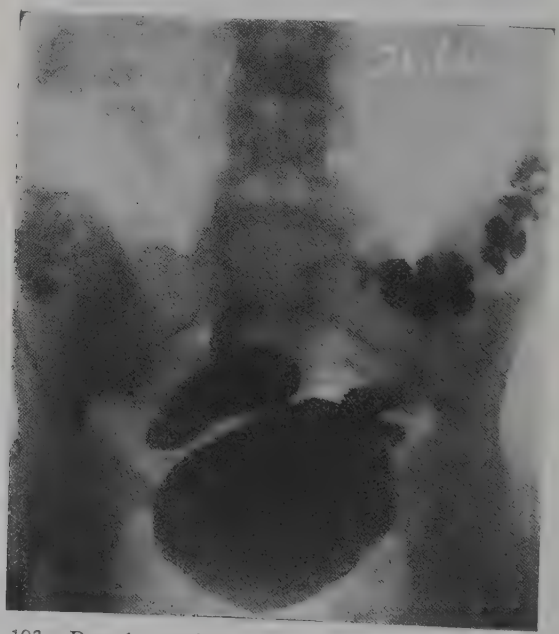


Fig. 103.—Rectal constipation due to rectal atony. (*Case.*)

Fig. 104.—Extreme degree of rectal constipation. 74 hours. (*Case.*)

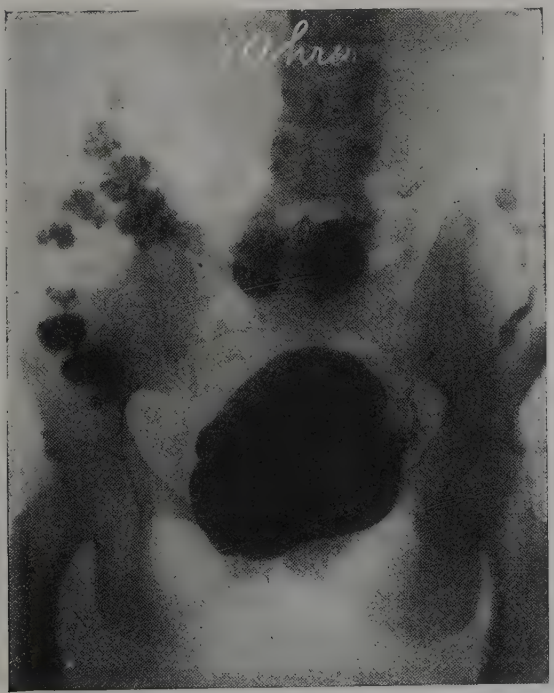


Fig. 105.—Extreme degree of rectal constipation. 50 hours. (*Case.*)

HYPERTROPHIED LEVATORES ANI MUSCLES.

Several instances of this have been met with in which the muscles become irritable or hypertrophied, and then clamp the rectum laterally, partially or completely blocking the passage. Many times they can be felt as thick, rigid bands at the sides of the rectum, about two inches above the anus, but in others they become irritable and spasmodically contract upon the introduction of the finger.

HYPERTROPHIED SPHINCTER MUSCLE.

Owing to the commonness of disease at the anal outlet the external sphincter is excited to frequent contractions, in consequence of which it often becomes irritable or hypertrophied and interferes with or prevents satisfactory defecation. This cause of constipation is rather well known among physicians and need not be dilated upon further.

COCCYGEAL DEVIATION.

According to Gant,[1] when the lower segments of bone project inward they cause rectal irritability and constipation and should be removed. Professionally I have seen quite a number of cases of forward displacement of the coccyx in whom constipation existed but I have never felt satisfied that removal of the coccyx was important for removing the constipation. Still it is possible that it might be a factor in some cases.

FOREIGN BODIES.

There is no doubt that foreign bodies, such as fish bones, may become lodged in the bowel or beneath the mucosa and cause constipation by obstructing the rectum; usually by exciting sphincteric contraction. Large bodies are readily removable by the finger or by use of the proctoscope with forceps.

ATONIC RECTAL CONSTIPATION.

One not uncommonly meets with voluminous rectum, largely atonic and sometimes having a caliber as wide as four or five inches. In such instances constipation usually exists. It is probably dependent in part upon the atony in the rectum and in that way interfering with the normal rectal reflexes which stimulate the desire and the act of defecation.

Lastly should be mentioned that there are rectal conditions in which constipation is a symptom. Among these anal fissures and hemorrhoids, particularly internal, are the most frequently observed.

DIAGNOSIS.

In the presence of a history of chronic constipation a search for a cause should be engaged in. This may be found in the life custom of the individual, his habits or his diet. If not, then a careful X-ray examination of the colon should always be made, and it is part of my routine, in the presence of a history of chronic constipation that an X-ray examination is invariably made. This may or may not disclose the cause, but it so commonly does so that it should be the routine. In the absence of any definite findings here a careful proctoscopic examination is called for, and when one conducts a practice along such lines in connection with this subject, very able work in the handling of constipated individuals is accomplishable.

While constipation might exist to a marked degree in some people without the production of any subjective or objective symptoms there are many in whom the daily movement of the bowels is essential to a state of well being. Quite a few people suffer immediately from subjective symptoms on the occasion of a degree of constipation, the lighter symptoms of which are sensations of fullness, distention and discomfort in the abdomen, at times referred to the stomach, loss of appetite, occasional belching, nausea, pyrosis and disagreeable taste, coated tongue, and headaches are often present, and with some people more or less colicky distress in the abdomen ensues. There may be an increase of indican in the urine and quite an elevation of temperature. In such individuals fecal masses may be palpable in the sigmoid region. As a rule, however, temperature is not present excepting when due to some complication such as inflammation, local peritonitis, or stercoral ulcer.

TREATMENT.

As was stated in the beginning of this chapter, constipation is a symptom, and thus the cause of the constipation stould be searched for and treated. With people who are habitually constipated with a movement every second or third day, and their general state of health good, it might be wiser not to engage in any method of treatment. But while the subject of chronic constipation has been overdone in its significance in medicine, it yet has an important bearing in the accomplishment of results possible by treatment, results which are favorable to the individual's health, and ofttimes most

beneficial. With persons in whom ignorance or false modesty are responsible for the constipation the importance of the individual's attention to the necessity of daily movements of the bowels and as quick a response as possible when the rectal reflex for evacuation has been instituted is called for. Of late the large number of hostleries met with in most large cities have caused people to be more apt to respond to these demands than was the case formerly.

The hygienic method of living, proper out-of-door life and exercise, and the diminution of strain and worry are often called for, and as few purgatives as possible should be employed.

Prousseau first advocated teaching regularity, which means that the patient should repair to a toilet at a regular time every day, preferably a short time after breakfast, and should remain sitting on the toilet, and after having been there five or ten minutes if there is no desire for the bowels to move, he may insert a glycerine suppository or take a small enema for the purpose. By persisting in this way, which is better than waiting three or four hours, a desire is stimulated and regularity may be cultivated, after which the use of the suppository or enema may be discontinued.

Diet.—Water should be taken in considerable quantity before breakfast and after meals through the day, and several times between meals. The use of high protein food, too much cane sugar or a diet too rich in carbohydrates or fats are to be avoided. The same may be said of milk, red wine, tea, chocolate and cocoa. The diet should be essentially vegetable; occasional taking of olive oil, from one to two ounces or more, has often an excellent effect. Quite a few people can keep their bowels regular by eating a raw apple in the evening before retiring. A sample of diet which is part of my routine is the following:

The plan of the diet is to eat three meals a day with nothing between times. The food should be of the normal kinds and simply cooked.

Drink at least five glasses of water a day, preferably before meals and between them, not during the meal. Any of the customary foods can be eaten but do not take more than two eggs in a day, and any of meat, fish and fowl but once a day, and this once in very small quantity.

Milk, tea or sour wines are forbidden, and no foods containing pits or seeds are allowed. Fruits, raw or cooked, should be eaten morning and evening; the liberal use of honey and the use of milk sugar instead of cane sugar on foods is advisable.

At breakfast each morning instead of a cereal, or with oatmeal, eat a handful of finely cut agar-agar with fresh cream. This can be purchased from the local druggist. During the course of the day eat one to three of the following gems, which should be baked twice a week:

Bran Gems: 2 cups of bran, 1 cup of flour, 1 cup of milk, ¾ cup of molasses, ½ teaspoonful of baking soda (dissolved in hot water), ½ teaspoonful of butter, ½ teaspoonful of lard, salt to taste. Bake in a slow oven 45 minutes.

If it is not possible to take the bran gems, take one or two tablespoonfuls of wheat bran cooked in milk with cream and sugar.

At dinner, or before retiring, take a dish of stewed prunes cooked in milk sugar or honey. It may be found that apple sauce sweetened with milk sugar will work as well as the prunes. If so, alternate this with the prunes, eating one on one day and the other the next.

At certain times each morning and evening make an effort to stool. This should be persisted in even if no success is obtained in the beginning. Its object is to establish a regularity in time. However, at any other time when the desire for stool comes on respond to it at the earliest possible moment.

Very often one has to write an individual diet to fit the individual condition of a person who is constipated. In this instance any of the above anti-constipation additions may be added to the diet, although the following serves admirably as an additional diet—that is anti-constipation additions which may be added to any diet:

Measures to encourage the bowels to move in a normal way are the following: Begin by taking three of the following bran gems during the course of the day, and if you cannot get them, take in place of them a good-sized dish of Pettijohn bran, or from 1 to 2 tablespoonfuls of wheat bran mixed with oatmeal or shredded wheat biscuit for breakfast.

Bran Gems: One-half teaspoonful of soda, saleratus, dissolved in ½ cup of hot water. Add, when dissolved, ¾ cup of molasses, then a tablespoonful of butter, salt to taste, 2 cups of wheat bran, 1 cup of bran meal, 1 cup of milk. Mix all the ingredients together, put in muffin pans and bake 45 minutes in a slow oven.

If the above does not move the bowels, retain the bran method which you are employing, and as an additional method take a dish of stewed prunes cooked with milk sugar or honey, or molasses, at the evening meal or before retiring.

If the foregoing two methods are not sufficient, take the prunes at the evening meal and a tablespoonful of petroleum jelly or vaseline, before retiring. With the institution of this take from 3 to 6 glasses of water during the course of the day, in the intervals between the meals and on arising and before retiring.

If then the bowels are not moving, add a few senna leaves to the prunes which you are taking, these being put in at the time of stewing.

If still the bowels do not move, drop the use of the bran, both in the biscuits and at breakfast, and substitute a handful or ½ oz. of finely cut agar-agar, eaten with cream and sugar at breakfast—this may be mixed with a cereal, but not cooked with it.

The foregoing methods you should follow by beginning with the first and adding one thing after the other until the desired result is accomplished. Do not become alarmed if the bowels do not move in the beginning, or if a day goes by during the time you are on the above additions.

The use of agar-agar has been resorted to by some practitioners for a considerable number of years for the relief of symptomatic constipation, and the question naturally arises as to the type of case in which it will prove most successful. It is in the case in which by increasing the bulk of the stool and rendering the mass less liable to contract into a firm cylinder the use of agar-agar answers to good purpose. When the stools are particularly dry so that they come away from the patient in hard, lumpy masses, the use of much fluid in the diet, and at times even a liquid diet, the drinking freely of water, and the use of agar-agar is particularly beneficial. Agar-agar may be used alone in a one-half ounce quantity in the morning, or it may be mixed with cascara sagrada as Adolph Schmidt suggested (Regulin), the same being taken at bedtime or twice a day after meals. When in a suitable case sufficient agar-agar is used, the stools are larger, softer, and come away without any colicky pains. Sometimes it is of value to use the agar-agar with jellies or marmalades. A very good practice is to use finely cut agar-agar with shredded wheat biscuit, taken in the morning. Another way to use agar is to cook it with a certain quantity of oatmeal, the same allowed to cool as jelly, cut into slices and eaten once or twice during the day with cream.

Einhorn[2] suggested the use of agar-agar mixed with various intestinal drugs, of which the following is a list:

1. Phenolphthalein-agar. Each level teaspoonful (1 gram) represents 0.03 gram (gr. ½) of phenolphthalein.

2. Rhubarb-agar. Each teaspoonful represents 1 cubic centimeter (ɱ16) fluidextractum rhei (U. S. P.).

3. Calumba-agar. Each teaspoonful represents 2 cubic centimeters (ɱ32) fluidextractum calumbæ (U. S. P.).

4. Gambir-agar. Each teaspoonful represents 2 cubic centimeters (ɱ32) tinctura gambir composita (U. S. P.).

5. Tannin-agar. Each teaspoonful represents 0.03 gram (gr. ½) of tannic acid.

6. Simaruba-agar. Each teaspoonful represents 1 cubic centimeter (ɱ16) tinctura simarubæ (U. S. P.).

7. Myrtill-agar. Each teaspoonful represents 1 cubic centimeter (ɱ16) tinctura myrtill (U. S. P.).

8. Ipecacuanha-agar. Each teaspoonful represents 1 cubic centimeter (m16) tinctura ipecacuanha (U. S. P.).

9. Sumbul-agar. Each teaspoonful represents 1 cubic centimeter (m16) fluidextractum sumbul (U. S. P.).

Bran which takes up water is indigestible and therefore it increases the roughage in the intestinal content and contains some intestinal stimulant and often serves to good purpose. It may be mixed with shredded wheat biscuit instead of agar, may be cooked in various forms of bread or cake, or it may be mixed with other cereals such as oatmeal or Pettijohn (Pettijohn bran). The following is a good formula for bran gems:

Bran Gems: ½ teaspoonful of soda, saleratus, dissolved in ½ cup of hot water. Add, when dissolved, ¾ cup of molasses. Then a tablespoonful of butter, salt to taste, 2 cups of wheat bran, 1 cup of bran meal, 1 cup of milk. Mix all the ingredients together, put in muffin pans and bake 45 minutes in a slow oven.

Baker's yeast, of which I have used Fleischmann's cakes, serves to excellent purpose for chronic constipation with some individuals. Why it is we do not know, but I have seen individuals whom no form of dieting served to regulate the bowels, there was no organic cause, and in whom the use of yeast, taking part of one cake at each meal established a regular movement of the bowels which had kept up for a number of weeks when the yeast was discontinued, and in several cases I am satisfied that the yeast cured the constipation.

For a long time various preparations of mineral oil have been in use for the alleviation of constipation, and while in some instances it does excellently, in the majority it is not to be recommended. Oftttimes the oil comes away without carrying any stool, and we are told that in this instance we are giving the oil in too large a quantity. It is often true that unless the quantity given is as large as is necessary to have the oil appear without feces, the results in overcoming the constipation are not efficient. Several years ago, even before Lane came out with his propaganda in which the use of mineral oil was a feature, I used the soft forms of paraffin, and I am satisfied that the results from the soft paraffins were better than with the today popular mineral oils. Paraffin is best employed in the form of petroleum jelly (vaseline) which is readily taken in the same way as mineral oil. The petroleum jelly can be warmed if desired to take it in fluid form. Some of my patients do not object to taking it when it is somewhat firm.

Lastly it should be mentioned that prunes cooked with any of the "dog sugars" are quite stimulating to peristalsis. For this

purpose milk sugar, glucose, or sweetening with honey or molasses answers to good purpose. Sometimes the cooking of a few senna leaves with the prunes is all that is necessary for just one addition to any diet to overcome a moderate degree of constipation.

Massage.—Various forms of massage have been engaged in to overcome constipation. Several of them are well worth while. Some individuals by stroking their abdomen when sitting with one hand from the caput coli to the hepatic flexure, then along the transverse colon, tends to stimulate a movement. For a long time the use of a three- or five- pound cannon ball rolled well over the abdomen and the coils of the colon and small intestine in a spiral manner, the patient in a dorsal position, was engaged in. I object to this, as well as all forms of mechanical massage which the individual uses himself, because it makes them too introspective and self- centered upon the condition. Vibratory massage with special attention paid to the sigmoid flexure, the seances lasting from about ten to fifteen minutes, two or three times a week is often of value.

Electricity.—Of considerable value is the use of the sinusoidal current with a large electrode fore and aft on the abdomen. Various other types of current have been employed, sometimes with much success, of which perhaps the next notable is the static current delivered in the Morton wave form.

Exercise.—In my opinion one of the most valuable methods of overcoming chronic constipation is the use of regular exercise. Of these there are none better than the U. S. Army setting-up exercises engaged in from ten to fifteen minutes each morning. Not a few individuals who have been to me have accomplished regular movement of the bowels by drinking one or two glasses of cold water on arising in the morning while engaging in the setting-up exercises.

Hirschmann believes that the direct stimulation of the atonic sigmoid and rectum by means of mechanical dilatation has, up to the present time, given the best results in cases of chronic constipation. He describes a simple pneumatic dilator for accomplishing this distention. It consists of a rubber bag with a stem, which is slipped over the distal end of a Wales bougie, No. 3-5, this bougie being canaled. Compressed air at a low pressure is allowed to enter the bag slowly, and distention to any desired extent is produced. A cut-off valve easily regulates the distention. Instead of the compressed air apparatus an ordinary atomizer bulb or a small bicycle pump may be used. The patient is placed in the Sims position for the treatment. This is repeated daily for from five days to a week. When defecation approaches the normal, treatments are given on alternate

days. The interval is gradually lengthened. He claims marked success from this method of treatment.

Enemata.—Enemata with hot saline solution at 110° to 120° F. for fifteen minutes three times a week alone, or combined with electricity (rectal method) or with the alternating cold douche, about 60° F., may prove of service in very obstinate cases. The soapsuds and water enema alone, using from one pint to one and one-half quarts, particularly when combined with about 4 ounces of glycerine, is of much value particularly when taken in the knee-chest position. This, however, is not a method of treatment excepting perhaps in the senile cases in which it may be engaged in about three or four times a week. Fleiner suggested a method of treatment which consisted of the pouring into the rectum at night before retiring from four to six ounces of olive oil, this being allowed to remain in during the night. By doing this a softening of the lower end of the feces takes place and usually a movement follows in the morning. My experience with olive oil is it is rather expensive and there is no special virtue to olive oil anyway, cottonseed oil answering to sufficient purpose.

In instances of fecal impaction with alarming symptoms the use of an enema consisting of equal parts of milk and molasses, the quantity being about a pint of each, serves to better purpose to stimulate the evacuation of the bowel than any other form of medicated enemata, even those containing Epsom salts, oxgall or turpentine.

The "high" enema is a misnomer, and should rarely, if ever, be attempted. Any one who has had experience with X-ray work knows that whatever fluid is put into the lower canal, even when there is considerable colonic spasm, in ten or fifteen minutes is in the right colon. In my experience enemata should not be much larger than a quart. A two-quart enema is a very large one and a three-quart enema exceeds the limit of safety.

Suppositories.—Quite a few individuals, particularly those in whom there is no rectal reflex, can be stimulated by the use of a glycerine or gluten suppository, or a small quantity of warm water containing one of these substances in solution.

In addition to the clysma treatment, the use of oil instillations into the rectum to overcome the spastic contractions of constipation, Soper[3] suggested the use of magnesium sulphate solution. His conclusions were the following:

1. Spastic contractures of the lower colon are etiological factors in many cases of chronic constipation.

2. These contractures are the result of disturbances in Meltzer's law of contrary innervation.

3. A saturated solution of magnesium sulphate applied locally to the contracted segment produces a relaxation. Repeated applications finally overcome the spasticity and permit the restoration of normal colonic function.

4. Contractures in the rectum and lower sigmoid, with accompanying dilatation of the colon, are found in many cases of post-operative abdominal distention. Magnesium sulphate enemata are very efficacious in relaxing the contractures and thereby relieving the distention and "gas pains."

5. Enemata of magnesium sulphate are also very useful in partial organic obstructions in the rectum and lower colon, inasmuch as they relax accompanying muscular contractures without stimulating peristalsis.

6. Magnesium sulphate solution applied by means of the cotton applicator greatly facilitates the introduction of the sigmoidoscope.

Medication.—It is my rule to use only the most mild laxatives and then they are used only in a temporary way. For the spastic form with fecal impaction belladonna may be of great value, this given as a tincture in large doses, from 10 to 15 minims and pushed to three or four times a day so that physiological symptoms are apparent.

Among the mildest laxatives are fluid extract of cascara sagrada, the aromatic fluid extract being the most pleasant to take. This may be taken in doses of from 4.0 to 8.0 grams, or the dried extract in from 0.065 to 0.324. Phenolphthalein in doses of from 1 to 5 grains (0.0652-0.324) at bedtime, or even T. I. D. may justify a trial. In my belief the combination that comes nearer to a physiological laxative is the following tablet which goes by my name:

Ext. cascara sag.	2 gr.
Podophyllin	½₂ gr.
Ext. belladonna	⅛ gr.
Strychnia sulph.	¹⁄₁₀₀ gr.

Sig.: Take one or two at bedtime.

The dose of cascara sagrada is just enough to be a mild intestinal stimulant; the podophyllin is far short of drastic, and it will be noted that the dose of extract of belladonna is small, simply enough to relax whatever spasms there may be, the dose of strychnia sulphate is small so as to act as a mild intestinal stimulant. I have used the above tablets for a number of years in individual cases and believe that they are as strong as anyone has any right to permit an individual to employ in a steady way, although I am opposed even to them,

and rarely use them excepting for one or two doses; excepting perhaps in the senile case.

The author feels that it serves to no advantage to include a long list of various purgative tablets or pills, most of which are well known to all members of the profession. It may be said, though, that rhubarb is an excellent drug and that the various saline purgatives which are resorbed by the stomach and upper intestinal canal and excreted by the sigmoid and upper rectal mucous membrane, may be necessary for a brief period. Jalap belongs to the stronger remedies, the same being true of castor oil and calomel, the latter two being excellent for occasional use.

Surgery.—It must be remembered by the reader that the writer believes that constipation is due to some error and that he is very conservative in all matters of surgery. Surgery is never permitted in his cases unless there is a definite diagnosis of something seriously wrong, and to which constipation is merely one of the symptoms. When surgery is indicated it is not for the purpose of relieving the constipation, but for the purpose of obviating or alleviating if possible some affliction in the abdomen or rectum. Then he is not at all in favor of Lane's teachings which, while helpful in a few instances, are erroneous in by far the greatest majority. Such a thing as doing colectomies, partial or complete, because of obstinate constipation or auto-intoxication is almost like beheading an individual to cure a headache.

I believe that there are instances of acute flexures or angulations of the colon or sigmoid which are important in the causation of constipation and fecal retention. The term acute is not used in a clinical sense but rather in a geometrical. I have seen a shortening of the mesentery by inflammation or adhesion, or the fixation of the gut by adhesions cause angulation which would narrow and even obliterate the intestinal caliber. Such conditions are sometimes congenital in which there is always a history of colic attended afterward by constipation and a congestion. One does meet instances where to remove constipation repair of defects about the flexures with perhaps a marked ptosis of the transverse colon, or an actual angulation of the flexures necessitates operative relief. Marked redundency of the sigmoid flexure also, and especially where there are years of progressive constipation, may give rise to symptoms varying from exaggerated constipation to actual obstruction, although never strangulation, and not a few of these cases are best relieved by operation. Several cases in which a suspension of the sigmoid flexure, transverse colon, or sigmoid was performed have improved markedly.

However, when engaged in operative procedure for the purpose of relieving constipation one is embarking on a course the result of which is liable to be questionable. The reason for this is there are so many other causes for the constipation than perhaps a redundent or angulated gut could cause that even when the latter is benefited striking results are not brought about. Developmental anomalies is certainly at the bottom of many cases of chronic constipation, and ptosis is often a factor. The best results from surgery are in those cases in which there is a recent acquirement of an active factor. It must be remembered that in the various suspension operations that while valuable in properly directed cases, they are disappointing in the remainder. At best these fixation points are unstable. Of course where marked adhesions binding down the gut or other definite factors like that exist, constipation as well as all the other symptoms, are most benefited by surgery.

Surgery may be called for in the various rectal causes of constipation. When the irritability of hypertrophied sphincters exists, the irritability of the muscle is due to ulceration or catarrhal inflammation, and much can be done by the application of hot fomentations to the lower abdomen and the daily injection into the bowel of hot oil containing bismuth, or a solution of hydrastis, boric acid, etc. When the interval of spasm is of frequent occurrence it can often be controlled by small doses of belladonna alone or in combination with opium. Where the O'Bierne's valves are hypertrophied to such an extent that the bowel is considerably occluded their dilatation is indicated. This can be done by introducing a large proctoscope up to the obstructive point and then passing a Wales's bougie of proper size through it. Divulsion can also be satisfactorily accomplished by tamponing with a rubber bag distended with air or water and left in as long as may be required. In a few cases surgical intervention is imperative, the obstruction being relieved by colostomy, resection of the sphincter, or the making of an anastomosis between the sigmoid above and the rectum below. When the Houston's valves are markedly thickened and rigid nothing short of a division will effect a cure. This is easily done by the use of cutting clips which divides the valves by pressure necrosis. Usually the valvotomy is followed by an immediate relief. It may be said that if the relief is not complete after doing the valvotomy alone and resort to the employment of diet, massage, electric or vibratory treatment are called for, the operation had not been indicated. I have had two cases of hypertrophied levatores ani muscles requiring operation, and in both instances the severing of the attachment of the muscles from

the bony coccyx brought about a cure. Gant has reported a number of such cases, some of which I have seen, and the results of the cure of the constipation have warranted the operation.

Sometimes in hypertrophied sphincter muscles a divulsion or dividing of the muscle is necessary. The divulsion may be accomplished gradually by the aid of anal dilators or bougies, or quickly and forcibly by thoroughly stretching the sphincter with the thumbs or index fingers. Forcible divulsion can be performed under either local or general anesthesia, of which nitrous oxide gas serves to good purpose. There is no objection to producing a temporary paralysis of the muscles by stretching, providing care is taken to avoid a rupture of the sphincter, an accident always followed by irreparable incontinence.

If in coccygeal deviation it can be concluded that the projection of the bone causes rectal irritability and constipation its removal may be necessary. Of course all foreign bodies should be removed, and atonic rectal constipation may be treated by injections of cold water or the slipping into the rectum of small bits of ice, the use of vibration, electrical currents known to strengthen the bowel musculature, by tamponing the rectum, and other procedures. It is my opinion though, once an atonic rectum, if you can judge by the non-diminution in the caliber of the rectum, no method of treatment answers any curative purpose.

References.

[1] Gant, Samuel Goodwin: N. Y. Med. Jour., April 15, 1911.
[2] Einhorn, Max: "On Agar as a Vehicle in Intestinal Therapeutics."
[3] Soper, Horace W.: Am. Jour. Med. Sci., Aug., 1918, No. 2, vol. clvi, p. 205.

CHAPTER XVII.

Nervous Disorders of Intestines.

General Remarks: In presenting the section on neurological conditions affecting the intestines the author feels himself at a disadvantage. This is due to several factors. There is no doubt that in the realm of neuroses, neurasthenia and psychasthenia, much pertaining to the abdomen is important, and there is further no doubt that the practitioner in this field who is a good student of human nature and neurology, as well as gastroenterology, has from a neurological standpoint much data and information in the understanding of functional abdominal conditions. At the same time there is the danger, if one studies neurology from the neurologist's standpoint, that certain theoretical conceptions and methods of treatment are engaged in as a result of over-enthusiasm, and therefore unwarranted from a clinical standpoint. Taking it from the general clinical standpoint on the other hand, if he is not abreast with more or less knowledge and familiarity with neurological conditions, ofttimes he will go astray in assuming certain symptoms as due to organic causes, and thus he will be deficient in rendering full service to some patients. Then again, on the diagnostic and therapeutic neurological side so much has been written, and is believed, on the importance of vagotonic and sympatheticotonic states and factors of endocrinology, matters that pertain to mental defect, inheritance, irregularities of the Mendel law, the various views such as Vimswanzer's, Freud's, Janep's, Jelliffe's, Savill's, White's, and the physiological theories, that from a clinical standpoint one has great difficulty in separating the grain from the chaff. The pendulum in medicine usually swings enthusiasticward. The surgeon views things from a surgical standpoint, the medical man from a medical standpoint, and the neurologist usually from a neurological standpoint, any or all of which may be incomplete in the individual case. There is such a maze in this entire subject that the attendant is often at wits' end, even though he may have had considerable neurological experience, to know just what to do. In cases in which there are organic and other factors complicated with neurological ones, when these are sent to neurologists one commonly has the experience that the neurologist views the entire picture from a neurological standpoint and gives advice which is sorrowfully

deficient in the way of rendering definite and substantial service to the individual. On the other hand, the clinical practitioner rarely has the time, the acumen or tendency of mind to "wetnurse" a neurotic, psychasthenic or neurasthenic individual. The neurological side has importance in clinical medicine, and the author will make an effort to present it in as practical a way as he is capable of, which he feels will be far short of what is required for a general and complete understanding of the subject. To the clinician, the suggestion that something is neurologically wrong, the patient being out of balance with himself or the world comes when after careful search no definite reason for the symptomatology can be elicited, and the patient in his manner and way hints certain suspicions to the attendant. I desire here to state for justice to the patient, that one should carefully scrutinize every possibility of physical error before considering the case neurological. Only in that way can we obviate the mistakes that neurologists make as frequently as anyone, and do the fullest justice to the case on hand.

NEUROSES.

A neurosis is a mental disorder, disturbing the personality and ofttimes bringing on most grievous forms of bodily suffering, pain, suffocation, etc. and is due to a pathogenic disturbance in the mind. There is generally a history of dreams, mental anguish, a sense of depression, and a close study of the individuals show that they are quite out of joint with the world and with themselves, especially so because they feel that those around them have no proper conception of just how ill they are.

It comprises one of the most widely spread of disorders, the greatest proportion of the cases never coming under medical inspection. I have known instances of obsession which has existed for many years, unknown even to the relatives about the individual. When one considers all of the persons in which are seen social maladjustments, inadaptability, inefficiency, inhibition, incapacity to meet necessary situations, abnormal fear of death or of poverty, hopelessness or even despair, and so on, it must be plain that many neurotics are constantly about us. It has been stated that strictly speaking neuroses are not diseases in the medical sense at all, but only in the social sense, and that neuroses are a result of a conflict between the individual and society, whereas other diseases are the result of a conflict between the individual and nature.

It is now known that to understand the neuroses best one must have some understanding of the psychoses. A distinction between

the neuroses and psychoses is made in the way that in the neuroses proper the pathogenic agents are operative at the actual time when the symptoms are manifested, whereas in the psychoses, or psychoneuroses, they are psychogenetically determined in the way that they have a definite meaning, expressible only in mental terms. In this distinction between the two there are so many grades which border on the one or the other that it is rare that differentiation can be made of as sharp demarkation. The two groups pass one into the other, into sub-groups and generalizations.

NEURASTHENIA.
(Actual Neuroses.)

Up to rather recently almost all cases of functional mind derangement have been classed as neurasthenia. The neurologists claim that this is unwise and not truthful as to the subject. The tendency now is to classify neurasthenia as an actual neurosis, and in that way sharply limiting it. This is explained on the fact that many cases are incorrectly diagnosticated as neurasthenia from a non-appreciation of the fact that psychoses are often to be seen in practice in a mild form, and that the greater number of cases thought to be neurasthenic really belong to the various forms of neurosis, which in etiology, pathology, and consequent treatment are quite distinct from this. They mention anxiety neurosis, anxiety-hysteria, and obsessional neurosis and suggest that the term neurasthenia be used as an enfeeblement or fatigue neurosis, the cardinal symptoms of which are mental and physical fatigue, difficulty in concentration of attention and application to work, sense of pressure in the head, irritable spine, various paresthesias, certain gastrointestinal symptoms, usually of the flatulent type, and so on. Therefore neurasthenia is now designated as a primary fatigue neurosis, and this is more common in the male sex, particularly among those of the intellectual class.

It is unlikely that heredity plays an important part in its production, although one does see families in which the neurotic feature is prominently examplified in more or less of the children, and in which one or both parents may be of that type. There is no doubt in individuals who have an unstability of their nervous systems that various infectious states, such as influenza, typhoid fever, and other infectious diseases in which there is a toxic agent, are distinct factors in its production, or at least the intensification of the symptoms. In the practice of gastroenterology, meeting with all sorts and kinds of individuals, affected in various ways, usually more or less chronically,

with symptoms referable to the gastro-intestinal tract, it is not uncommon, when a condition has existed in which the subjective symptoms are pronounced or perhaps made more pronounced by the mental attitude of the patient, that eventually the individual becomes neurotic and even distinctly neurasthenic. The commonest in the production of these are chronic states of intestinal toxemia, and at least a third of my cases have had experience with neurologists or sanitariums of various kinds.

I am inclined to believe that mental strain and overwork is only one of the factors in the bringing out of symptoms in susceptible individuals. Where the general condition of the body is functionally and organically well, mental strain and overwork if sensibly engaged are not commonly factors of importance in the bringing on of these conditions. On the other hand, onanism, if only mental, has to do with the production of these states, and there is no question that in gastro-intestinal practice, it is a factor of some importance in the control of unexplainable symptoms.

Treatment.—As painful as it may be to engage in it, conversation must be had to find out whether auto-erotic habits, sexual in kind are present. If so, advice must be given that these be replaced by normal sexual functioning or abstinence.

Whenever mechanical, functional, or organic derangement of the body can be diagnosticated, these require cardinal attention. During the time of doing this, hope, encouragement, the winning of confidence, inspiration of the individual, and all these factors which the average medical man unconsciously engages in are at his command. The condition may be so marked that rest from work and strain, the removal of the individual from the family, all sources of worry or excitement, perhaps a change of occupation, the instilling of agreeable interests, the use of electricity, building up the general health, perhaps recourse to the rest-food method of treatment or that of Weir Mitchell would be required. In studying cases that have clinical, functional, or organic derangement in whom the neurotic and neurasthenic element is pronounced, what characterizes them is an up and down course to end betterment, and in a little while one must take them in hand and deal with them gently or firmly as may be required, make a few new suggestions and set the premises for the time when the next depression sets in. In accomplishing this the assistance of a well-meaning, honest, and well-trained neurologist, may be called for while some definite condition is being treated, because it must be remembered that the end of many of these individuals, if not skillfully handled, is that of chronic, nervous invalidism.

PSYCHONEUROSES.

The psychoneuroses are best understood in the term hysteria, in which our former ideas of hysteria must be modified to include minor types of the disorder as well as those that are well known.

It is well to remember that the apparently unreasonable reaction, behavior and attitude of the hysteric when properly investigated usually proves to be fully intelligible and well-grounded. Such individuals react differently from the normal, sometimes being afraid of objects, situations or occurrences that excite no fear in the normal, and on the other hand, they may be strikingly unafraid of matters that normally excite anxiety. This sort of condition is due to want of balance, disturbance of mental equilibrium, and instability that is so often applied to them.

Since these individuals have types of mind which rebound back to worse things in the past rather than the more minor condition of the present, they usually magnify whatever symptoms there are present by their own imagination adding to it. One commonly has the experience of, say, a psychasthenic individual, in whom constipation and a marked meteorism existed in whom by some simple means, such as the instillation of oil in the rectum at night, regular movements have been brought about, this individual constantly magnifies as time goes on the marvelous cure you have brought about in her condition. In such a person not uncommonly, whatever may be the gastrointestinal malady that had been acquired, the instillation of oil into the rectum at night could be a cure-all for whatever might be wrong.

Treatment.—It is not the purpose of the author, nor is he qualified, to enter into matters of mental analyses or psychotherapeutic methods of treatment. It is his belief that what is not accomplishable by the Weir Mitchell treatment is only less so by any of the other measures. To the original theory of Weir Mitchell, that the central nerve cells were starved of nutrition and excess feeding was required, rest is added to bring about the best results. While the latter is a factor of importance, it allows also the suggestion possible of being brought about, mentally in the individual when in bed, and by the suggestive exercises of the physician on his visits. The hypodermic use of cacodylate of strychnia and glycerophosphate, and the steady use of the malt preparations add much to permanency of results.

In the ambulatory case, benefit is brought about by handling patients gently but firmly, seeing them frequently enough so that they think you have an interest in their case, guiding and advising

them on various matters of life, the care of themselves, and their attitude toward others, and having them feel a complete confidence in you as well as you being a haven to which they can come with their woes and troubles.

The author has entered somewhat into a description of these conditions, because relatively the number of cases he sees of abdominal conditions which are plainly due only to neuroses, neurasthenic or psychasthenic states are few, but he knows that with others they may be common, and that in many of the clinical functional and organic disorders more or less of these states do exist, and unless one is conversant with that side, the fullest justice to these individuals may not be accomplishable. The medical profession must always remember that in these people, whatever the malady may be and however slight it may be, they always imagine that it is far worse than it is, and unless he handles them with this consideration, they may become the adherents of any of the fads and fancies of therapeutics religious and otherwise that go to make up the vagaries of what by misnomer some call an advanced civilization. There is a very large body of apparently normal individuals who are seeking relief from conditions which are resident in disturbances in the emotional sphere of the brain, and these flitter along through the fads, new creeds and fancies, getting solace for the time being and getting renewed solace from a new one when they have become satiated with an old.

VAGOTONIA.

In this connection it may be advisable to present briefly some remarks on the condition of disturbance of the vegetable nervous system which Eppinger and Hess have drawn attention to. There is no doubt in my mind that a diagnosis of vagotonia is sometimes warrantable, perhaps more preferable than that of neurosis. It must be remembered that there are certain definite symptoms making possible the diagnosis of vagotonia in man.

Relaxation of the zone of Zinn, or stimulation of the ciliary muscles is evoked by the activity in the autonomic system. When present, it causes a sphericity of the lens. It is best discoverable when tonus due to a strong autonomic stimulant can overcome the paralytic effect upon the iris due to atropine. It is notable that in vagotonic individuals a mild spasm of convergence, a kind of convergent strabismus, has not infrequently been observed. In these people there is usually an increased salivation which is marked after pilocarpine and uncontrolled by atropine. It is believed that the type of

heart which shows a bradycardia which changes into tachycardia under the influence of atropine must be considered as a typical manifestation of increased vagus tone. Eppinger and Hess laid considerable stress on bronchial asthma due to a spasm of the bronchial musculature and being relieved by atropine and adrenalin as due to an over-irritable state of the vagus.

The stomach phenomena of the vagotonic is rather well understood. There is a distinct effect upon the tone, peristalsis and secretion, all being enhanced, and usually controlled by either atropine or the suprarenal preparations.

In the intestine very little is known of the subject of vagotonia. The functional test of the duodenum and ileum are very difficult and can only be followed radiologically, and the secretory and absorption activity is ofttimes obscure, usually complicated by the compensatory action of the colon. It is believed that autonomic stimuli will cause an increase of peristaltic action in the small intestine, and this I have seen present in a number of instances. This condition may bring about both a diarrhea and constipation, which when one is in doubt is always very much enhanced by the use of pilocarpin and relieved by atropine or adrenalin. The spastic type of constipation which affects more particularly the left side of the colon may be to some extent vagotonic in nature. In my opinion, it is more often caused by a status of saccharo-butyric toxemia causing an irritative spasm in a local way. However, vagotonia may cause the condition, and in such cases it is not uncommon that a condition of colitis and membranous enteritis also exists.

Like the clear-cut neurological conditions, vagotonia may be met with in combination with other diseases, and in that sense may require attention as part of the treatment.

CHAPTER XVIII.

Intestinal Parasitic Conditions.

General Remarks: With the exception of tapeworms and some intestinal round worms, very little was known of intestinal parasites until 1838 when Dubini of Italy discovered the hook worm, the blood fluke and the dwarf tapeworm as noted by Bilharz in Egypt in 1851, and the Chinese human liver fluke by MacConnell in India and MacGregor in Mauritius in 1874. The first parasitic protozoön to be discovered and recognized as such was the ciliate Balantidium coli as a cause of dysentery, which was discovered by Malinsten in 1856. In 1875 Lösch discovered the dysentery ameba. Other parasites affecting the blood stream and other parts of the human economy were discovered at various times, but these will not concern us in the work on the intestines. Only those which are intestinal in habitat will be considered.

The effects of parasites on their host are almost as numerous and as varied as are the kinds of parasites, and vary beside with the susceptibility of the individual concerned, his physical condition, and complication with other infections. It may be said that the parasitic infections damage their host in one or more of three ways. (1) By robbing it of foods which have not yet been assimilated and utilized; (2) by mechanically injuring the tissues or organs; (3) by the formation of excretions or toxins which act as poison. Of these three, the second and the third are the most important. There is no doubt that a natural immunity exists in some people against parasitic infection, usually exemplified in one or more. The power of the blood in vertebrate animals to react against invading organisms or poison by producing substances which will destroy them is one of the most wonderful adaptations in all the realms of nature, and has no doubt been acquired by the vicissitudes which man has passed through in the many generations he has populated the globe. Nevertheless, in so far as intestinal parasitology is concerned, numerous are the cases of infection. In my own practice, intestinal parasites were met with in 6 per cent. of 5000 private patients, and 11 per cent. of 5000 clinic patients. The reason for the disproportion here is that among the clinic patients are a number from the rural districts of our country, Europe and Asia, whereas the private patients represented more

particularly people of much better financial means and city dwellers. There is no doubt that among those who live close to the soil the incident of intestinal parasite condition is very much enhanced. Smithies[1] reports that among his cases of the Middle West, protozoa were met with in 93 instances out of 1000 stool analyses, and regarding this he gives the following summary:

Cases of Protozoic Enterocolitis in Three Years.

Patients with intermittent or chronic diarrhea	86
Patients without diarrhea	7
Total cases	93
Patients from south of Springfield, Ill.	4
Patients who had visited in the South	11
Patients who had never been in the South	78
Patients from small towns, rural communities or farms	70
City or large town dwellers	23
Patients who had habitually drunken well or shallow river water	78
Patients whose water supply was seemingly good	15
Patients who were large eaters of fresh garden truck, bananas, of unwashed fresh fruits	66

Sistrunk[2] reports that in the Northwest among patients who had never been in southern countries in the feces of 145 patients—35 of which were used as controls of the function of the pancreas, leaving 110—there were 65 instances of intestinal parasites.

Lyon[3] reports that in the stools from 163 individuals, 72, or 44 per cent., were found to harbor one or more kinds of parasites. Rosenberger, also from the East, reports that in 1280 specimens of stool 92 contained ova or parasites. There are no good statistics from the Tropics or South, which is unfortunate, because in these climates the incident of intestinal parasites is far more numerous than in the North. Roche[4] reporting from the Salonica war area states that in the examination of stools of 893 cases of diarrhea and dysentery occurring among the troops of the Salonica area during the months of August, September and October, 1916, the following were met with: Non-pathogenic amebæ were found in 81 cases, and amebæ which were not identified because of lack of time in 39 cases. Entameba histolytica was found in 37 cases; Entameba minuta in 47 cases. Flagellate protozoa were found in 217 cases divided as follows: Lamblia intestinalis, 73; Tetramitus mesnili, 90; Trichomonas, 45; Cercomonas, 9. Coccidia were found in 18 cases.

A perusal of past literature shows that while the larger organisms, like the intestinal worms, have long been regarded as highly detrimental, and lately much has been learned of the hookworm

infection, there is still a debate going on as to the noxiousness of other forms of parasites. Numerous papers on the pathogenicity or non-pathogenicity have appeared in which such parasites as the Cercomonas, Trichomonas, Lamblia, Megastoma, Balantidium coli are debated upon. The non-pathogenicity articles were the rule some years back, the tendency being to consider them as pathogenic in the papers of better quality and the work more intensive, and today it may be said that in this whole field of work many of these affections which have gradually been coming under suspicion have now an accumulating mass of evidence proving conclusively that the parasites are commonly pathogenic to man. The experience of trained parasitologists in the Tropics where alimentary protozoan infections of the body are more common, points distinctly to the importance of more intensive study, not only of the possibilities in this field, but an enlargement of general medical knowledge on the subject. This is true also in the more temperate zones where the largest number of the population of the world live, and one needs only to be a practical student of these conditions to soon become aware that almost any of these parasites are inimical to normal conditions in the intestinal canal.

It is a well-known fact that many of these parasites exist in multiples of two or more. Sistrunk presents the following which is fairly accurate with my experience in the East. He reports from his 65 cases the following:

Patients showing:

Entameba tetragena	16
Entameba coli	11
Entameba histolytica	9
Small ameba and unclassified ameba	12
Trichomonas intestinalis	32
Cercomonas intestinalis	9
Lamblia intestinalis	4
Strongyloides intestinalis	1
Balantidium coli	1
Ascaris lumbricoides	1
Trichocephalus dispar	1

Patients showing the following combinations of parasites:

E. histolytica and E. tetragena	1
E. histolytica and Lamblia intestinalis	1
E. histolytica and Cercomonas intestinalis	1
E. histolytica and Strongyloides intestinalis	1
E. tetragena and small ameba	1
E. tetragena and Trichomonas intestinalis	6

E. tetragena, E. coli, and Trichomonas intestinalis	2
E. tetragena, Cercomonas intestinalis and Trichomonas intestinalis	1
E. tetragena, E. coli, Cercomonas intestinalis and Trichomonas intestinalis	1
E. tetragena, Trichomonas intestinalis, Lamblia intestinalis and Ascaris lumbricoides	1
E. coli and Trichomonas intestinalis	3
E. coli, small ameba, Trichomonas intestinalis and Cercomonas intestinalis	1
Trichomonas intestinalis and Cercomonas intestinalis	2
Trichomonas intestinalis and Lamblia intestinalis	1

The following table shows the number of times each species was found in the patients who had never been in the South or Tropics:

E. coli	6
E. histolytica	1
E. tetragena	11
Trichomonas intestinalis	17
Balantidium coli	1
Lamblia intestinalis	3
Ascaris lumbricoides	1
Unclassified amebæ	9
Cercomonas intestinalis	3

Smithies reporting the coincident infections met with in his cases presented the following:

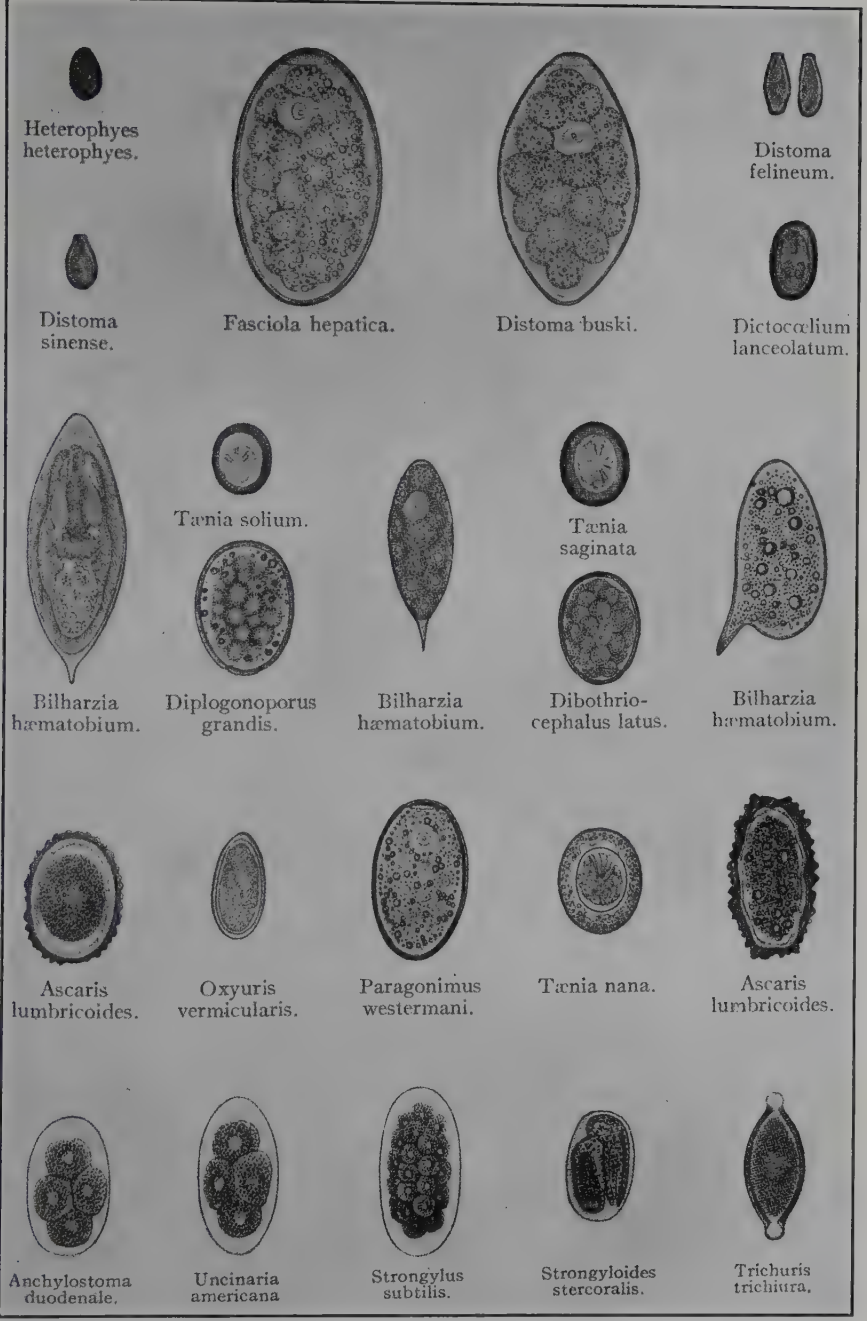
Coincident Infections.

Cercomonas with entameba	12
Trichomonas with entameba	14
Cercomonas, trichomonas and entameba	4
Trichomonas and cercomonas	8
Cercomonas and lamblia	3
Cercomonas, trichomonas and lamblia	2
Trichomonas, cercomonas and megastoma	1
Trichomonas and balantidium	2

It therefore is important to remember that the treatment of many of these cases is for more than one of the types of parasites present.

The intestinal organisms which affect the human intestinal canal are divisible into four classes of protozoa. First are those of the ameba family, of which the most important is the Ameba histolytica; second, from a pathogenic point of view, stand the ciliate balantidium coli, which, however, is not common in most parts of the world; third, the simple bi-flagellate form such as Bodo, Cercomonas and Prowazekia, some of which are probably only accidentally parasitic; and fourth, the highly organized multi-flagellate form such as Trichom-

PLATE LX



Drawings of eggs of intestinal parasites. (*Cabot.*)

onas and Giardia which are very common human parasites and are of clinical significance.

All the protozoa which make their home in the digestive tract of animals resemble one another. Nearly all of them secrete for

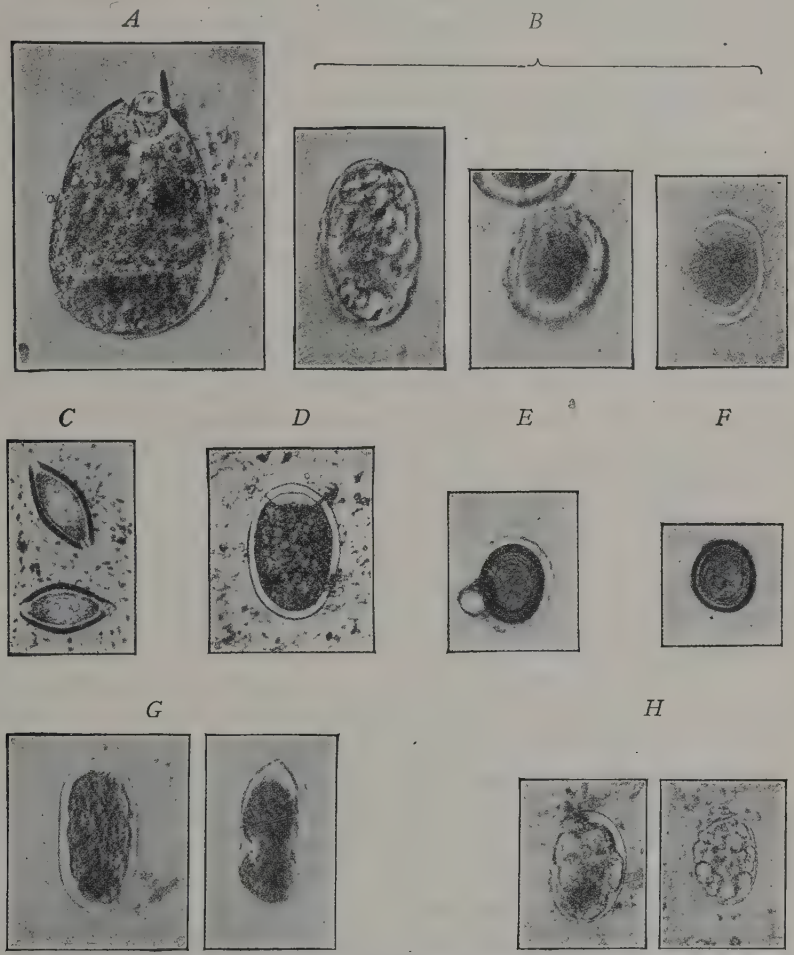


Fig. 106.—Eggs of intestinal parasites (after *Cabot*) all are magnified 250 diameters. *A*, Distoma buski. *B*, Ascaris lumbricoides. *C*, Trichuris trichiura. *D*, Dibothriocephalus latus. *E*, Tenia solium. *F*, Tenia saginata. *G*, Uncinaria americana. *H*, Anchylostoma duodenale.

themselves resistant transparent cysts which protect them from drying up or from the presence of an unfavorable medium. In this encysted state, intestinal protozoans are able to exist under the unfavorable conditions found outside the body of the host, and are

capable of remaining in this state in a sort of torpid condition for long periods of time until they gain access to a new host. This encysted state follows closely the general biologic law necessary for their protection and perpetuation. The cysts of the intestinal protozoan are analogous to the resistant eggs of intestinal worms, and like worm eggs, their presence in the feces of infected persons serves as an easy means of diagnosis.

One of the evidences that intestinal parasites excrete toxin is the presence of eosinophilia supposed to be for the purpose of destroying toxins of the blood, yet some of the leucocytes are apparently for the purpose of gathering and destroying bacteria or other foreign cells. An increased number of eosinophiles is therefore sufficient reason for assuming the presence of toxins for them to destroy, thus they have diagnostic significance. The normal number of eosinophiles varies from 1 per cent. to 4 per cent. of the total number of leucocytes, and in infections with such parasites as trichine, blood flukes, ecchinococcus cysts, etc., nearly always there is a rising to 5 per cent. or higher, and in some cases this reaches over 70 per cent.

In addition to the presence of eggs in the feces and the eosiniphilia, the following are the most prominent symptoms met with: Diarrhea, abdominal pain, distress in the digestive organs, loss of weight, achylia gastrica, anemia, blood in the stools, and fever and chills. These are the general symptoms found rather commonly through all of the intestinal infections, the special and more classical ones connected with individual infection will be mentioned in the subdivisions that follow.

THE AMEBA.

It is essential to realize that amebic infection of the intestine, amebic colitis, cannot be studied from amebic manifestations in other parts of the body. For this reason the term, Amebiasis has been employed. In the human intestinal canal mainly two forms of entameba are met with, the Entameba coli, and the Entameba histolytica.

The Entameba coli is of fairly world-wide distribution and found by Schaudinn in 50 per cent. of healthy individuals in West Prussia, and Vedder found it in 72 per cent. of Filippino scouts in the Philippines. In no cases in which it has occurred alone had the carrier suffered from dysentery, nor is it possible to cause dysentery symptoms in lower animals by infection with this organism. The Entameba coli in the active ameboid stage measures about 22 to 38μ, although it may be smaller. Under ordinary circumstances the ectoplasm is not well differentiated from the endoplasm except when

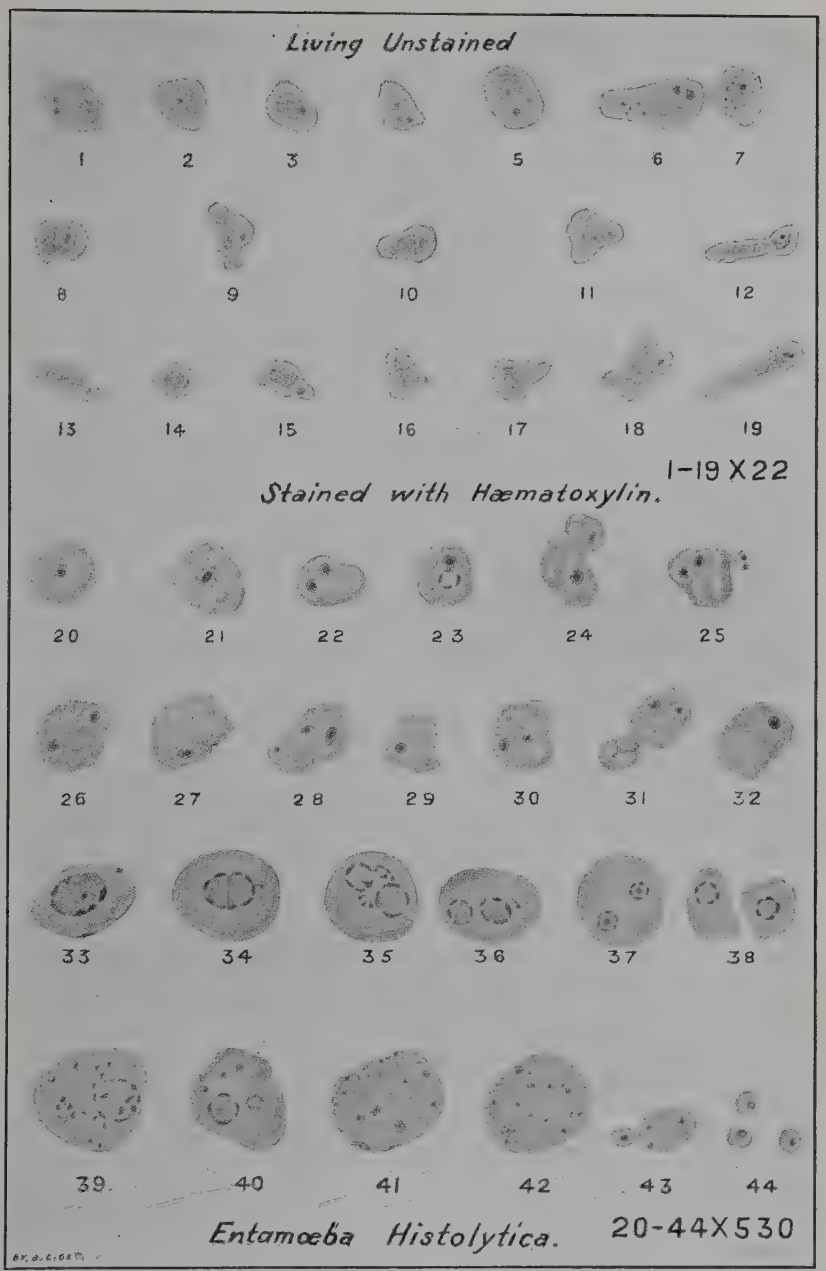
pseudopodia are being extruded. The cytoplasm is coarsely vacuolated and it often contains starch grains, bacteria and other bodies. Red blood corpuscles are but rarely seen within it even when present in large numbers in the feces. The vacuoles do not pulsate. If stained with Lugol's solution a glycogen reaction may be seen. The nucleus is eccentric, though in active specimens it may be more central. As a rule it contaains an abundance of chromatin peripherally arranged, giving the appearance of a well-marked nuclear membrane. In the living state this ameba is more porcellaneous in appearance and less active than the histolytica. It reproduces by simple division. The fully formed cysts measure from 16 to 25 μ. They are larger than the cysts of the Entameba histolytica, and they have a well-marked double-contoured cyst wall. The encysted stage is found in solid feces, the amebic stage in diarrheal motions or after the administration of a purgative. Emetine does not cause their disappearance, nor do degenerative forms occur after its use as is the case in the amebic stage of Entameba histolytica.

The Entameba histolytica is found in the mucus passed in the acute stage of amebic dysentery, and in the body lesions produced by its invasion of the tissues. Its size is from 25 to 30 μ, but in the dividing stage it might be larger. It shows fairly active movement with the protrusion of pseudopodia, and in a typical specimen, the ectoplasm is well developed and easily distinguishable from the endoplasm, though at times all distinction may be lost. The endoplasm is fairly homogeneous and not distinctly vacuolated, and may contain red blood corpuscles. Ordinary food particles are usually absent and it gives occasionally a glycogen reaction with Lugol's solution, although not often. The nucleus in the living organism is often only seen with difficulty. The limiting membrane of the nucleus is thin or absent, the chromatin is arranged peripherally, and it is scanty, the granules being fairly uniform in size. Sometimes the chromatin is massed into more or less irregular clumps. The nuclear membrane is continued within the nucleus as the lining network of fibers upon which some very fine granules are seen. At the center of the nucleus a granule can usually be distinguished, the karyosome, surrounded by a clear area limited by a ring of granules which represent the inner limit of the lining network. The next state in the life-history of Entameba histolytica is found in soft diarrheal stools, and it is probably a saprophytic and preencysted stage. It is smaller than the fully developed ameba just described. There is no definite ectoplasm, the endoplasm contains vacuoles. Red blood corpuscles are usually absent. Starch grains

and bacteria may be present. Chromidia are frequently seen in the cytoplasm, and the cytoplasm in this often shows a well-marked glycogen reaction. This stage is more resistent than the histolytica stage. The resting, or encysted stage, is found in the solid feces in the intervals between attacks of dysentery, or in altogether latent cases in which attacks of dysentery have been entirely wanting. It is the most easily recognized form of the Entameba. The minuta form is frequently associated with it. The cysts have an average diameter of about 11 to 14 μ and are therefore smaller than the Entameba coli cysts. Occasionally cysts of 19 μ diameter may be seen. The cyst wall is not so thick nor so well defined as the cyst wall of the Entameba coli. At times it may be only definitely seen when the contents contract. Chromidial bodies are more constantly present.

Examination of the Feces for Amebæ and Their Differential Diagnosis.—The material may be examined fresh, or it may be fixed and stained. Each method has its advantage. In the fresh specimens the live ameba may be seen moving, but for a thorough study of the organism, fixed and stained specimens are necessary. Further, in the acute stage of dysentery in which blood-stained slime is passed, the parasite is found in the active stage, but Entameba histolytica is most easily distinguished from Entameba coli in the encysted form. This is seen in the solid feces, and to obtain it no purgative should be administered. In soft diarrheal motions and after purgatives, the organism appears in the minute form, and this is very hard to distinguish from the Entameba coli. In fixation it is most important that the specimen should not be allowed to dry. Slime is simply spread on a cover-slip and fixed by floating the slip on the fixing solution, the best one being an alcohol sublimate solution. In this it is allowed to remain from 5 to 15 minutes, then washed in distilled water, stained in either alum hematoxylin, if a rapid result be required, or in iron hematoxylin if full cytological details be needed. It is again washed, dehydrated in increasing strengths of alcohol, cleared in xylol and mounted in xylol balsam.

Having obtained the specimen, examination should be begun with low power. A warm stage is not necessary except in cold weather. Ameba and Entameba may exhibit movements. It then becomes necessary to distinguish between them. The ordinary saprophytic amebæ, such as limax, are small, with a nucleus containing a large central karyosome surrounded by a small quantity of peripheral chromatin, the vacuole being contractile. Their cysts are rare, with a thick double contoured membrane often polygonal, and a single



Various forms of Entameba histolytica. 1 to 7. Living unstained amebæ, some containing red corpuscles. 8 to 19. Shapes successively assumed by an ameba during its movements. 20 to 32. Amebæ fixed in alcohol and perchloride of mercury and stained with hematoxylin. 33 to 38. The same showing simple division. 39 to 44. The reproduction of Entameba histolytica by budding off of young forms. 1 to 19 under Zeiss D, Oc. 2×220. 20 to 44 under 1/12 inch, Oc. 2×530. (*Rogers.*)

nucleus hard to distinguish. The ameboid stage of flagellates is also characterized by its smallness and a large karyosome in the nucleus. The size of the Entameba is important; 20 to 30 μ. This distinguishes the resting ameba from pus-cells, epithelial cells, etc., and if the nucleus can be seen, its relatively small size and ring-shaped arrangement of chromatin distinguishes it from all bodies excepting a large epithelioid cell sometimes seen in mucous stools. The following is a short table of the differences between Entameba histolytica and Entameba coli:

AMEBOID STAGE.

	Entameba Histolytica.	*Entameba Coli.*
Size	25 to 30	22 to 38 μ
Motility	Active, lasting some hours.	Sluggish.
Nucleus	Relatively thin nuclear membrane, poverty of chromatin.	Nucleus often like a heavy ring.
Contents	Red blood corpuscles common.	Red blood corpuscles very rare.

The minuta stage occurs in diarrheal stools, and is hard to distinguish from Entameba coli.

ENCYSTED STAGE.

	Entameba Histolytica.	*Entameba Coli.*
Size	11 to 14 μ	16 to 25 μ
Nuclei	Typically four, usually same size.	Typically eight, may vary in size.
Cyst wall	Thin.	Thick, and double-contoured.

It must be remembered that sometimes the parasite is not seen at the first examination, as its excretion may be intermittent, and therefore, if necessary, two or three examinations should be made. Further, after an acute attack of dysentery, for a week or two, the ameba may appear to be absent, and then cysts begin to appear. A mixed infection may also be seen of Entameba histolytica and Entameba coli. It is not uncommon to find other parasites and cysts such as Balantidium, Lamblia, Trichomonas, and other flagellates.

The behavior of Entameba histolytica outside the body and its reaction to chemicals and drugs must now be considered in order to elucidate as fully as possible the propagation and treatment of the disease. The ameboid active stage is very vulnerable. It cannot withstand desiccation, dying within a few hours outside the body. The study is best accomplished by means of the iron hematoxylin

method of Heidenhain. The Wright method is also valuable in differentiating the feces of intestinal amebæ. Giemas's stain will also give very good results. The characteristic changes in the protoplasm are best brought out by Wright's stain, with which in the case of the Entameba coli the endoplasm becomes a dark blue, while the ectoplasm is light blue. In the case of the Entameba histolytica this is reversed, the endoplasm being light and the ectoplasm a dark blue. The Entameba tetragena, on the other hand, does not stain well with Wright's method. In each case the chromatin of the nucleus stains red, the tint being lighter in the Entameba histolytica than with Entameba coli or tetragena. With the hematoxylin method, the chromatin stains a very dark black, while the protoplasm takes a light grey coloration, and this method is most valuable for studying the characters of the nuclei and their changes during the reproductive stage. In the first place, in the active stages of the organisms the following differences are found in the nuclei of the three human species. In the Entameba coli the chromatin is abundant, forming a well-defined nuclear membrane with marked thickened portions projecting from its inner surface, while in the center is a large mass of chromatin, the karyosome. In contrast with the Entameba coli, in the case of the Entameba histolytica the nucleus is a much less prominent body with comparatively little chromatin, without a well-developed nuclear membrane, and with a much smaller central karyosome. The nucleus of the Entameba tetragena closely resembles that of Entameba coli in having a well-developed chromatin nuclear membrane and a large karyosome.

The following is a table of differences between the human intestinal amebæ and those of other forms, as given by Rogers:

Pathology.—The earliest lesions are small red raised dots, which may be a little larger than a pin's head. They are produced by congestion or even hemorrhage into the mucous membrane over the early exudation into the submucous coat beneath. They are well seen in the ascending and transverse colon. As soon as a slightly larger size is reached, a light yellow spot appears in the centre of the darkly congested area, due to loss of the epithelial portion of the mucous membrane allowing the gelatinous, citron-yellow colored infiltration of the submucous coat to appear on the surface, thus producing the earliest stage of ulceration. At this time the most marked feature is the elevation of the affected patches, causing them to stand up like small buttons from the healthy mucous membrane. Sometimes these lesions present an oval form, with the long axis across that of the bowel. The next stage is an extension of the lesions across the long axis of the bowel, the invading ameba meeting with the

I. Morphological Characters in Unstained Specimens.

	Entameba Coli.	*Entameba Histolytica.*	*Entameba Tetragena.*
Size	10 to 30 micromillimeters.	10 to 70 micromillimeters.	10 to 50 micromillimeters.
Nucleus	Well defined, in center of endoplasm.	Not well defined, at junction of endo-ectoplasm.	Well defined.
Protoplasm	Granular throughout and not clearly differentiated into endo- and ectoplasm.	Endoplasm granular, ectoplasm clear and well differentiated from endoplasm.	Like E. histolytica, but clear ectoplasm less well developed.
Color	Always gray.	Often of a greenish tinge.	Often of a greenish tinge.
Vacuoles	Few, non-contractile.	More numerous, non-contractile.	Like E. histolytica.
Pseudopodia	Small, finely granular, and not well differentiated from endoplasm.	Large, clear, and well differentiated from endoplasm.	Clear and well defined, but smaller than in E. histolytica.
Movement	Very sluggish.	Very active.	Very active.
Contents	Very few or no red corpuscles.	Often with numerous red corpuscles.	Often with numerous red corpuscles.

II. Morphological Characters in Stained Specimens.

	Entameba Coli.	*Entameba Histolytica.*	*Entameba Tetragena.*
Nucleus	Large amount of chromogen, well developed nuclear membrane.	Small amount of chromogen, badly developed nuclear membrane.	Large amount of chromogen, well developed nuclear membrane.
Protoplasm with Wright's stain	Endoplasm dark blue. Ectoplasm light blue.	Endoplasm light blue. Ectoplasm dark blue.	Not well differentiated.
Reproduction	Nucleus first divided into 8, and afterward the protoplasm divided to form an equal number of young amebæ.	Nuclear chromogen dispersed in protoplasm and young forms containing parts of it bud off.	Nucleus divided into 4, and an equal number of young amebæ formed.
Multiplication	By simple division.	By simple division.	By simple division.

least resistance along the course of the blood-vessels encircling the gut in the submucous membrane. Once the ulcers have extended round the greater part of the bowel in this manner, through extension of the exudation into the submucous coat leading to necrosis of the overlying mucous membrane by cutting off its blood-supply and by pressure, the production of the most advanced lesions is only a matter of degree.

Advanced lesions found in the large gut show a greatly thickened bowel wall, being easily felt through the abdominal wall as a sausage-shaped mass, and evincing tenderness due to commencing local peritonitis. Perhaps considerable gangrene would exist in a fatal case. Then masses of enormously thickened raised patches of tawny, yellow ulceration are present. In severe cases there may be black cobweb-like sloughs of mucous membrane, produced by the rapid extension of the exudation into the submucous coat, destroying the vitality of large areas of the undermined epithelial layers and causing them to be cast off as necrosed portions.

In the gangrenous form of the disease, the lesions are no longer confined to the mucous and submucous coats, but the gelatinous infiltration extends to the muscular layers, separating the fibers and causing a softening of the tissues with the final involvement of the peritoneum, occasionally with rupture. In the more chronic lesion in which there has been a process of healing, the ileum is usually healthy, while the cecum shows a large shaggy, yellow ulcerated patch of the acute disease. In the ascending colon there may be an area of yellow patches with more or less narrowing here and there. There may be evidences of puckering of the mucous membrane around large chronic ulcers or at the site of completely healed ones. In these cases, secondary cirrhosis of the liver may not infrequently be met with.

A microscopic examination of the early lesion shows a section through a small raised amebic ulcer with the typical tawny yellow center, the change noted being an extensive small-celled infiltration of the submucous coat, which raises the mucous membrane to form the button-like elevation on the inner surface of the bowel wall, and separates the muscular from the mucous layer. The mucous membrane itself is comparatively little damaged as compared with the lesions in bacillary dysentery. These lesions in character and extent form the seriousness of the infection.

The commonest complication in amebic disease is abscess of the liver. This may be one large abscess or a number of small ones. Sometimes these abscesses are met with in cases in which there have been little or no dysenteric symptoms during life, but in which,

nevertheless, an Entameba histolytica infection exists. The liver is generally in an acute hepatitis with enlargement, the blood-vessels en-

Fig. 107.—Section through an early amebic ulcer of the colon. (*Rogers.*)

gorged, and the tissue rather soft and pliable before the abscess formation. In the fully-formed abscess there is an irregular cavity,

often partially divided up by bands or trabeculæ, and containing a soft, creamy liquid material, which is usually colored reddish brown and looks like anchovy sauce. It may, however, be yellowish. The contents are not true pus, but consist of liquefied liver tissue, mixed with red blood corpuscles, some leucocytes, sometimes small masses of necrosed liver tissue, ameba, and occasionally Charcot Leyden crystals.

The lung is sometimes affected secondarily to a liver abscess, but in care cases an abscess may occur in the lung without there having been a previous abscess in the liver. Then, too, a liver abscess may rupture through into the lung cavity. These pulmonary abscesses are irregular in form. The lung around the cavity is usually consolidated, and edematous fluid may be squeezed out on pressure. Occasionally there is no definite abscess cavity, the lung at the site of the lesion appearing as a boggy infiltrated mass of tissue.

Amebic infection of the brain occurs as a rare manifestation. When this formation is present it is usually found in the cerebrum, any part of which may be affected. The dura mater may be engorged with blood, but it is rarely adherent to the structures beneath, except by fine filamentous adhesions. The condition of the pia mater depends on the situation of the abscess, which if it be superficial, the pia mater may be greyish, reddish or even violet in appearance.

Other rare manifestations have been recorded, such as parotitis, in which the amebæ have been demonstrated, abscess of the spleen, cystitis, infections of the female generative organs such as salpingitis, infection of the skin, etc. It must be remembered with this disease that a veritable amebemia may occur, and with lapse of time further amebic manifestations may be recorded.

Symptomatology.—Cases of intestinal amebiasis differ greatly in severity from a clinical point of view. Thus at one end of the picture we have ulceration, and even extensive lesions, without any signs or symptoms, and at the other end we find severe gangrenous lesions with sloughing of the whole thickness of the gut and perforation into the peritoneal cavity. The course and symptoms are therefore found to differ greatly. A classification has been made by Phillips,[5] as follows:

(a) Acute intestinal amebiasis (amebic dysentery).
    (1) Simple acute, of moderate intensity.
    (2) Gangrenous dysentery.
(b) Chronic intestinal amebiasis.
    (1) With symptoms.
    (2) Latent.

In addition to these varieties, we must recognize the carrier cases in which there need be no actual lesion present. The carriers may be contact carriers who have never suffered from dysentery, and convalescent carriers who have had the disease, but continue to harbor the parasite.

SIMPLE ACUTE DYSENTERY.

The onset of this common form may be sudden, with pain in the abdomen of a colicky nature, which is soon followed by discomfort and bearing down at the anus. The amount of the tenesmus, however, varies, being more severe when the lesions extend to the rctum. In dysenteric ulcers limited to the cecum and ascending colon tenesmus would be absent. The pains would then be felt in the abdomen, and at the same time there might be vomiting. Diarrhea sets in quickly. At the commencement the feces may be the ordinary contents of the bowel, but they change in nature, becoming watery, with an admixture of mucus, and may entirely lose their ordinary character. The appearance of mucus in the stools is accompanied or followed by blood, the amount varying from a slight tinging of the mucus to almost pure blood in some cases. The stools are of great frequency, and may vary from 12 to 20 or 30 in 24 hours. Usually the quantity of stool is not much more than a drachm. Fever is usually absent, or, if present, it is of moderate degree. It runs no definite course, and all acute cases usually show it at some time. If untreated, a condition of this sort may last several days, or a week or two, then it may gradually subside or it may become chronic, the patient suffering from time to time from attacks of dysenteric diarrhea, or acute exacerbations may occur, forming definite relapses.

The onset is not always so acute. It may begin as an ordinary diarrhea, which is thought at first to be of a simple nature, but in the course of a few days it develops into a typical dysentery. In some of these mild acute cases there is probably only a superficial catarrhal ulceration, not unlike that seen in mild cases of bacillary dysentery. Some of the acute cases, however, because of the constant distress, the vomiting together with developing emaciation, dry and inelastic skin, as in cases of cholera, retracted abdomen, may show an extreme degree of illness. After convalescence is established, the pains disappear, the stools lose their blood and become more fecal, the mucus which exists a little longer gradually disappears also. After a bad attack convalescence is usually slow, the patient having become anemic from the loss of blood and malnutrition.

GANGRENOUS DYSENTERY.

This usually commences in the same way as an attack of the simple type, but the patient rapidly gets worse, the abdominal pain increases, and the temperature may rise higher. The tenesmus also increases. The stools usually become blood-stained very quickly, very offensive, and take on the appearance of scrapings of raw, putrid meat. In other cases they become greenish, brownish, or blackish, and definite sloughs appear as tough, stringy masses. As the gangrene increases the stools may become less frequent, the temperature falls and becomes subnormal, the pulse becomes very frequent and thready, rapid emaciation sets in from the pain, vomiting and septic absorption, the abdomen becomes retracted and very tender over the colon, cold sweats appear and the patient dies from exhaustion. In some cases perforation occurs, and then the symptoms of peritonitis make their appearance. In other cases, however, the sloughs separate, the patient takes a turn for the better, and slowly regains health and strength.

CHRONIC INTESTINAL AMEBIASIS.

Conditions of this type may be divided into two forms, that with symptoms, and the latent type.

With Symptoms.—A patient suffering from the chronic form of the disease with symptoms complains of recurrent attacks of diarrhea, in which from time to time blood and mucus may be passed with some pain and tenesmus. The pain is usually a dull ache rather than an actual colic, and may be referred to the epigastrium and so simulate dyspepsia. Many such patients will give a history of preceding acute dysentery which has simply become chronic, but in others the disease has been chronic from the commencement. The general health suffers, and emaciation is not uncommon, and with the abdominal discomfort there is also frequently some actual dyspepsia. The result is that the patient looks thin, sallow, a little careworn, and is never really free from pain. The action of the bowels may be rather frequent, but the patient is not sufficiently ill to take his bed. This condition may last for months or years, and in some cases stricture of the bowel may result. In others, the symptoms may be those of a troublesome recurrent diarrhea with the necessary passage of blood or mucus alternating with constipation, and the nature of the diarrhea is only apparent on the discovery of the ameba.

Latent Type.—In the latent form of the disease there may have been a preceding acute dysentery, which is thought to have been

entirely cured, but post-mortem examination would supply evidence of extensive ulceration. Or there may be no history of dysentery, and it is well known that an amebic liver abscess may arise when there has been no history of an antecedent dysentery, and yet the gut may be the seat of amebic ulceration. These ulcers are sometimes revealed by sigmoidoscopic examination. Some of these patients, however, complain of an aching in the abdomen at night and in the early morning. Flatulence occasionally occurs; there is also occasional constipation, not relieved by purgatives, or purgation sometimes produces an excessive action. Neurasthenic symptoms are not uncommon, with perspiration of the hands and feet, and in many cases there may be no loss of weight. The only positive fact which can make the diagnosis certain is the presence of the ameba in the encysted tetragena stage.

General.—According to Rogers,[6] three-quarters of the cases of amebic dysentery show a leucocytosis, and he also states that when the number of leucocytes exceed twenty-five thousand the case is serious and requires energetic treatment.

Diagnosis.—The systematic examination of the dejecta is important in which Goodeve's method of washing the stools is worthy of being followed. The lower bowel should be examined by the sigmoidoscope. In some cases with ulceration lower down ulcers may be discovered, but in chronic amebic dysentery it is rare for ulcers to be situated low down in the sigmoid flexure or the rectum. In bilharzial disease it is the lower part of the large intestine and the rectum that are chiefly involved, and, as a means of differential diagnosis, the sigmoidoscope is of value.

In the differential diagnosis, the non-dysenteric conditions must first of all be excluded. In children the possibility of an intussusception should be borne in mind, but with the adult, malignant disease of the bowel is the most important disease to be excluded. Syphilitic ulceration of the rectum will also be revealed by the sigmoidoscope. Mucous colitis may create some difficulty in diagnosis, but the microscopic examination of the feces for ameba or other protozoa and of the blood for agglutination reactions, will exclude one or other of the dysenteries. To distinguish between the dysenteries themselves, in amebic dysentery and in balantidial and flagellate dysentery, though the onset may be sudden, there is usually little or no fever, whereas in bacillary dysentery, the sudden onset is usually accompanied by fever which may be high. Amebic dysentery, moreover, runs a less typical course than bacillary dysentery. Bacillary dysentery tends to occur in epidemics, whereas amebic dysentery is endemic.

The careful examination of the feces will reveal protozoal parasites in protozoal dysenteries, and an examination of the blood or cultivation of the stools will reveal bacillary dysentery. Further, there is the therapeutic test by emetine, this drug tending to clear up amebic dysentery but having no effect in bacillary dysentery. In bilharzial dysentery, which is almost invariably a chronic disease, the presence of the ova in the feces, and probably also in the urine, and the occurrence of lesions in the lower bowel, confirm the diagnosis. It must be remembered, however, that mixed infections occur. A patient harboring amebæ may be attacked by bacillary dysentery. Bilharziasis and amebiasis may occur together. Lastly it should be stated that other organs in the body should be examined in all cases of amebiasis.

Treatment.—Ipecacuanha is the drug which is historically associated with the disease, and which to this day is the most efficient drug, though for many years it was under a cloud. Its use was introduced with failure in cases of bacillary dysentery, the reason being that no differentiation from the amebic form was then possible. Attention was then paid to the alkaloids which had been isolated under the name of emetine and which was really a mixture of the three alkaloids, emetine, cephalin and psychotrine. For about 50 years, from the days of Bardsley who used emetine with distinct success until the days of Walsh, from 1842 to 1891, this substance had not been used. Vedder then drew attention to the strong amebicidal properties of the alkaloid in very weak dilutions on living amebæ. Probably to Rogers more than to anyone else is due the credit of popularizing this form of treatment. He shows that in emetine we have one of the most powerful specific drugs known, being probably more efficient in amebiasis than quinine is in malaria. But, as in all protozoal infections in which a specific drug is known, prolonged use is necessary to effect a complete sterilization of the body from the parasite, a result seldom achieved. The other alkaloids are not so efficient, and cephalein is more emetic, so whenever possible pure emetine is to be recommended. Therefore the emetine hydrochloride is in most frequent use today.

A patient suffering from amebic dysentery should be confined to bed and warmly clad, especially around the abdomen. If pain and tenesmus be extreme a dose of morphia may be administered. Hot applications or fomentations may be applied to the abdomen. If he has not already taken a dose of castor oil one should be given. The diet should consist entirely of liquids, and if there is vomiting, it may be simply barley water or oatmeal water, albumen water or some form of broth. In any case it is well to avoid milk for the first

twenty-four hours. Alcohol in any form should also be avoided. If necessary, hypodermic injections of strychnine or camphor may be administered if there is any tendency to collapse or heart failure. While waiting for a definite microscopic or bacteriological diagnosis, a hypodermic injection of emetine, one grain (0.06 gram) should be given. There is no danger from this, and if the case is one of bacillary dysentery, no harm is done, while much valuable time may be saved. In ameba infections this treatment should be followed daily. In a favorable case the stools rapidly become normal, and in three or four days the patient is apparently cured. The emetine may now be continued in daily doses of half a grain. After the first injection of one grain, it is preferable to give it in half-grain doses night and morning. The use of the drug should be continued for ten days, and then given in interrupted course of a week for several weeks in the attempt to rid the patient completely of the parasite. The use of the emetine is supposed to kill off the parasites that are actually in the tissues, but not the encysted form in the gut. If, however, all the active amebæ are killed off as the cysts develop, the majority of the cysts may eventually be destroyed or expelled from the bowel, unless there is some resting-place that we do not know at present. The injection should be made into the deeper tissues or into a muscle. Emetine can be given by mouth, or in keratin or salol-coated pills. It is fairly effective so given, but may cause some nausea for the first dose or two, then tolerance is developed. It should be given at night on an empty stomach.

My experience with the above method, or with the injection of emetine hydrochloride has not been uniformly successful. It has been my observation that following the biologic law of all forms of low animal life that whenever there is danger of their destruction they go into the encysted form. It has been stated that the encysted form is only found in the content of the gut. But this I question, because it must be that the encysted form can take place within the confines of the tissue. What is of interest is that some cases even when thoroughly treated in the course of time are very prone to recur. It is my belief that the encysted form may be present in the tissues, or that an hypodermic injection of emetine arriving in the presence of the parasite may drive it into the protective, or encysted, stage. For that reason I am partial to the method used in the older days, not the infusion of ipecacuanha, but the one suggested by Simon, of giving ipecac in rather large doses at night. In this instance large quantities of ipecacuanha are present during the entire night in the intestinal canal, thereby soaking the tissues with the drug, which in

my belief, is a better method of treatment than to give emetine hypodermically, which in many instances becomes distributed throughout the entire body unnecessarily. Of course in the presence of an amebic infection in the lungs, brain, etc. the emetine hydrochloride would be the most efficient procedure, but where the disease complications can be excluded in the case, it is my belief that the intestinal method is superior. In this instance the patient is put to bed and given an initial dose of castor oil, no food being allowed for twenty-four hours. The first night 75 grains of ipecacuanha in 5 grain pills, salol-coated are given at one dose, and each night the dose is reduced by two pills, in the course of a week the treatment being over. Along about the second or third day food is allowed by mouth, this being of the fluid form, and after the course of treatment has been concluded semi-solid foods are allowed. Sometimes along early in the morning there is considerable nausea, in which instance a small dose of morphine or opium may be required to control the symptoms. Following out this plan of late years, I have not seen a return of the trouble, which was not so when I was using the emetine hydrochloride treatment. Mention should be made that when the results from the ipecacuanha method are not satisfactory, massive doses of bismuth should be employed, the dose for an adult being about a teaspoonful of bismuth subcarbonate or subnitrate three or four times a day. The results with this are often striking.

Local treatment of the bowels is often of great value and relieves the tenesmus. The best solution to use is plain warm saline solution given slowly and not under any pressure, by choice out of a douche can. This empties the lower bowel often giving relief for several hours and it may be repeated night and morning. Some advise a weak solution of quinine—1 in 1000 for its amebicidal value, but it is doubtful if this is really necessary during the emetine or ipecacuanha treatment. In the later stages when the blood has disappeared but mucus remains, daily enemata of tannin 1 in 100 to which quinine in the proportion of 1 in 1000 may be added is of material service. Tannin is also amebicidal. During convalescence, the diet is to be gradually increased and care is to be taken to ensure a daily action of the bowels, a saline being used for the purpose if necessary. Under such treatment most cases get well rapidly. Sometimes it is wise to use the organic salts of silver, such as protargol, a 5 per cent. solution.

In some bad cases, however, surgical intervention may be necessary. This may take the form of appendicostomy or cecostomy followed by a washing of the bowel daily. In these instances a lavage with collargol 1 in 500 twice daily is worth while. In the case

of stricture, a colotomy may be necessary if local treatment fails to relieve.

The patient usually requires tonic treatment in addition; iron, or iron and arsenic for the resulting anemia. He should also take care not to become constipated, using laxatives, such as liquid paraffin for the purpose. Care should be taken to avoid chills, especially of the abdomen. A flannel binder or cholera belt should be worn at night. It is best to have these individuals take a sojourn in some cold climate for a period after the course of treatment has been discontinued. In instances of abscess of the liver sometimes it is necessary to aspirate. It is a well known fact that these abscesses are sterile with regard to the ordinary bacteria of suppuration. The advantage of aspiration if successful, is obvious. The abscess does not become infected and the time and dressings saved are considerable. A fair-sized needle is used, and when the cavity is reached the pus is slowly aspirated so as to empty the cavity as thoroughly as possible. Rogers advised the injection of the cavity at the time and daily afterward, with a sterilized solution of quinine, 10 grains to the ounce, the quantity injected being 2 to 4 ounces. In many cases the aspiration may have to be repeated two or three times, and in some cases may even require the open operation. In all cases the use of emetine greatly adds to the chance of recovery. The occurrence of intestinal hemorrhage after the evacuation of a liver abscess, requires the use of calcium salts and the injection of a sterilized solution of gelatin. This should be about 10 per cent., and it must be a solution supplied by a reputable firm of manufacturers and guaranteed sterile, especially as regards the bacillus of tetanus and malignant edema.

Ameba of the lung is best treated by general anti-amebic treatment. If the abscess be purely one of the lung, rapid improvement is the rule. Even though the abscess has lasted some months, cure will result in most cases. Intrathoracic collections of pus such as occur when the abscess opens into the pleura and extends up beneath it are easily treated by rib resection and drainage. Abscess of the brain should be energetically treated with emetine.

CRAIGIASIS.

Closely related to amebic dysentery is a form of ameba described by Craig now called the Craigia hominis. Another species described by Barlow in natives of Honduras suggests that they are rather general in distribution, and probably all closely allied.

This organism differs from the ameba and resembles the flagellate in that it passes through a flagellated stage in which one long flagel-

late may be noticed. Resembling the typical ameba it is about one-half the size of the dysentery ameba, from 10 to 25 μ in diameter, and when moving exerts several blunt pseudopodia. It possesses a parabasal body in addition to the nucleus. The animal multiplies by simple division for a time, but eventually encysts, rotating on its axis during the process of forming the double-walled cyst. When in the encysted form they are considerably larger than those of the dysentery ameba (15 μ in diameter) and contain about 40 high refractive bodies which later escape from the cyst and develop into little flagellated organisms known as "swarmers." These grow to several times their

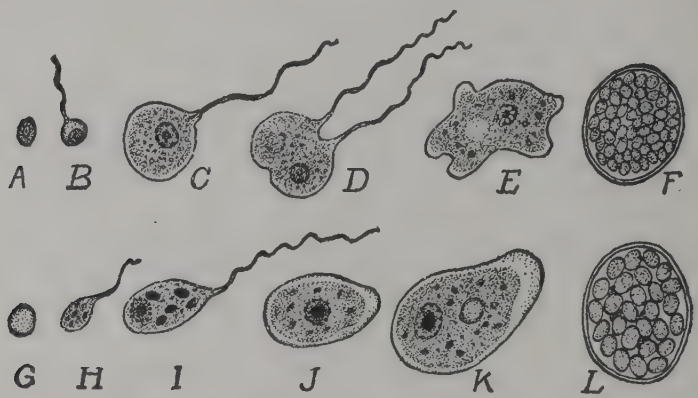


Fig. 108.—Life cycles of Craigia. (*Barlow.*)

original size, multiply a few times by simple division, and finally lose their flagellum and pass again into the ameboid stage.

Symptoms.—Barlow describes craigiasis as he found it in Honduras as more insidious than amebic dysentery and not so distressing in its early stages, but ultimately quite as dangerous a disease. The symptoms are those of a constant diarrhea with bloody and mucus stools, loss of appetite, abdominal pain, distention, all of the symptoms which are similar to those of amebic dysentery. In Barlow's experience, liver abscesses are even commoner in craigiasis than in amebic dysentery. In the communities in which it is found it is considered more dangerous than amebic dysentery because of the larger per cent. of healthy carriers who, though showing no marked symptoms for years, may be a constant means of spreading the infection which is believed to be from polluted water.

Treatment.—Emetine is destructive to craigia, as it is to other amebæ, answers as the best treatment, although it is reported that injections of the hydrochloride are not as effective as in amebic

dysentery, because the free-swimming flagellated forms escape. Complete and rapid cure is best effected by combined treatment with emetine injected into the blood and ipecac taken by mouth, accompanied by occasional flushing of the bowels with saline laxatives or enemata to remove the cysts. The same preventive measures used against amebic dysentery are applicable to craigiasis.

MOUTH AMEBÆ.

It has been known for a long time that amebæ are common denizens of the mouth. The type seen is known as the Entameba gingivalis (buccalis), but as to whether it is pathogenic and as to whether it is a cause of pyorrhea is now believed to be settled. It is considered harmless, and when met with pyorrhea, is merely incidental. However, whether the formation of the pus pockets in Rigg's disease is initiated by the ameba, or by other organisms, it is certain that the Entameba gingivalis is almost without exception found in the lesions and at the very bottom of them, often burrowing into the inflamed tissues to a depth of several times its own diameter, devouring cells and transporting bacteria.

Symptoms and Treatment.—The symptomatology of pyorrhea alveolaris need not be described here, it being well known. I desire simply to state that when this condition of the mouth exists it is best to refer the case to a dentist. There are not many dentists who seem to handle cases of pyorrhea in a curative way. All that pertains to prophylaxis of the mouth, and the mechanical side of dentistry is to be attended to, but many times the infection is deeply rooted in the peri-dental membrane and nothing worth while is accomplished by them alone. Fortunately of late the dental profession have paid more attention to this subject, and here and there men are springing up who are qualified to handle cases of Rigg's disease in the proper way.

The necessity of mouth hygiene and all that pertains to prophylaxis in the way of oral sepsis is important in connection with the prevention of this condition.

CILIATES.

(Balantidiasis. Ciliate Dysentery.)

This is a rare disease, but has been met with in Germany, Russia, the United States and the Philippine Islands. The Balantidium coli is met with in larger or small numbers in the stools, and in the fresh state is a large oval granular protoplasmic mass from 70 to 100 micromillimeters in length and 50 to 70 in breadth, showing

an oval nucleus anteriorly, together with contractile vacuoles and coarse granules of partly digested food. At the anterior end is a slight depression, the peristome, and all around the rest of the surface are cilia, which are longest near the peristome and slope backward from their attachment to the finely striated cuticle. Multiplication is chiefly by transverse division, other described methods of reproduction being still disputed. The parasite may also encyst, especially if kept outside the body under unfavorable conditions, when they quickly lose their motility and many of them degenerate and disappear. They are successfully cultivated and are readily killed by antiseptics and by a 1 in 5000 solution of hydrochloride of quinine.

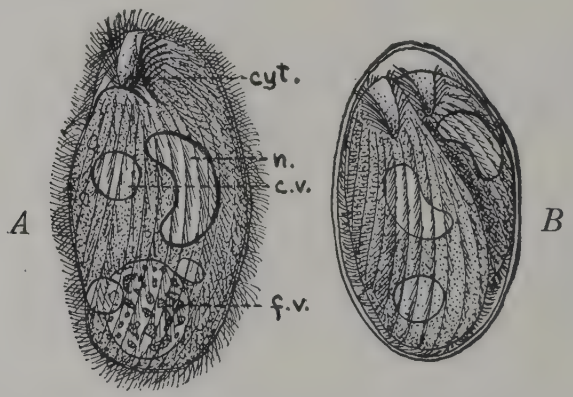


Fig. 109.—Balantidium coli. *A*, Free ciliate from intestine. *n*, Nucleus. *c v*, Contractile vacuoles. *f v*, Food vacuole. *cyt*, Cytostome. *B*, Cyst as passed in feces, containing two parasites. × about 500. (*Wenyon.*)

These organisms are found both between the tube glands of the mucous membrane and in the submucous layer, where they may enter the blood-vessels and be carried to the liver and set up abscess formation there. The typical lesions are produced in the large intestine, especially in its lower part, being commonly limited to the sigmoid and rectum. Congestion and catarrh with edematous swelling first appear, going on to produce deep round or oval ulcers covered with mucus, somewhat resembling those of the amebic disease, but containing numerous Balantidia coli if examined within a few hours of death. Sloughing and even perforation may result. There is generally no leucocytosis but the eosinophiles may be increased. Infection is believed to be through water, while healthy pigs often harbour the parasite and monkeys have been found to be naturally infected.

The Balantidium swims about in the contents of the large intestine, devouring particles of fecal material. As long as the animal

confines its activity to this, no ill effects result excepting perhaps a drying effect upon the mucous membrane. But it also has the power of invading the tissues and causing ulceration, perhaps after an injury from some other cause has given an opening for its invasion. Although most persons affected do not show any dysenteric symptoms, these are likely to appear at any time, and when they do appear they are of a very serious nature and cause a high mortality, bringing about a horrible condition of the large intestine with ulceration from end to end with shreds of mutilated or dead tissue hanging from the wall.

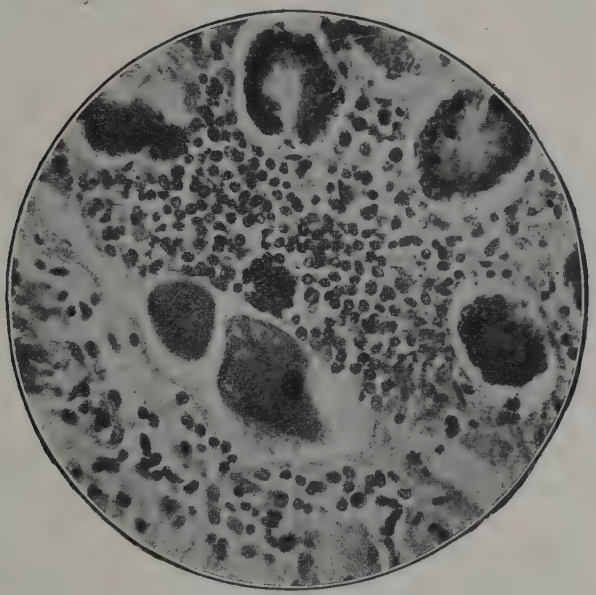


Fig. 110.—Two balantidia lying just beneath the mucosa. Numerous round cells, with beginning ulceration. (*Bowman.*)

The symptoms are said to resemble those of amebic dysentery, and in the early stages diarrhea may also be present, while later typical dysenteric stools are passed. The stools are alkaline, watery, or pea-soupy, they contain undigested food and mucus, they are not so frequent as in amebic dysentery, usually numbering from 6 to 15 in the 24 hours. As a rule they are somewhat small. Blood may or may not be present at first. Microscopically at this stage they contain mucus, food particles, many leucocytes, especially eosinophiles and the balantidium; enemata will usually bring away a further amount of feces. There is usually some distention of the abdomen, and it may be tender on pressure especially over the descending colon. The tongue is usually thickly coated with fur. As the disease pro-

gresses and ulceration spreads, blood appears in larger quantities in the stools. It may sometimes be in the form of a regular hemorrhage. With this there is progressive anemia, partly from the loss of blood and deficient nutrition, but probably partly also from the hemolysin secreted by the parasite. The skin becomes sallow and dirty-looking, and inelastic, and there may be irregular fever. Should perforation occur then signs of peritonitis arise, which usually ends in death. A sigmoidoscopic examination has in some cases revealed ulcers in the rectum, and in this way they may be observed to heal under treatment. A scraping from an ulcer generally yields many parasites. The blood changes are not constant. As a rule there is no leucocytosis. Eosinophilia may occur, but it is not always present. When there is much eosinophilia other worms are usually also present, as in Dutcher's case, in which he recorded 40 per cent. of eosinophiles; but many ankylostomata and other intestinal worms complicated the disease. In most cases, 1 to 2 per cent. seems to have been the usual percentage of eosinophiles. A continued hypoacidity or anacidity of the stomach frequently complicates the disease, and such a condition with dysenteric symptoms might aid the diagnosis. The diagnosis can only be established by finding the parasite in the discharges. The mortality of the disease is fairly high in advanced cases. Out of 111 cases collected by Strong 32 died of the disease, which gives a percentage of mortality of 29.

Treatment.—Various drugs have been tried such as calomel, saline, ipecacuanha and others. Thymol yielded good results, 4 to 5 grams being given in two hours. Under its use dead Balantidia are passed. Diarrhea may disappear and no relapses occur. Local treatment by large enemata of collargol or protargol or some other organic silver compound seems to be indicated, as they are very efficient balantidicides. Tannin, a 1 per cent. solution, may be used. If the Balantidia are destroyed by quinine solution, it would be worth a trial to use the quinine solution in a trans-intestinal lavage method, such easily being made with the bisulphate salt. Unfortunately there is no specific treatment for balantidial dysentery as for amebic disease. The removal of the Balantidium by trans-intestinal lavage with a hypertonic solution is easy, but when it has once invaded the walls of the gut, very little is accomplished.

The prevention of balantidial dysentery is not only in the sanitary disposal of human feces, but also in the care of hogs, since Balantidium is a common parasite of these animals, and is probably normally a hog parasite. A large proportion of hogs are affected in almost all warm and temperate countries, and it is almost always

in hog-raising countries and in places where men come in close association with them that balantidial dysentery occurs.

BI-FLAGELLATE PROTOZOA.

The most primitive and the least harmful of all of the intestinal flagellates are the bi-flagellated forms, several genera of which have been found in the human intestine. These are, namely, Bodo, Cercomonas and Prowazekia. The animals of this genus represent trypanosomes in the general form of the body and in the possession of a parabasal body and an undulating membrane, but have an additional free flagellum. In Cercomonas, according to Wenyon, the

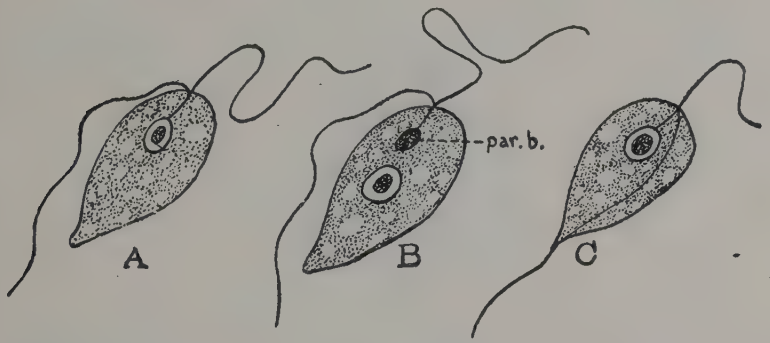


Fig. 111.—Bi-flagellated parasites. *A,* Bodo; note absence of parabasal body. *B,* Prowazekia; *par b,* Parabasal body. *C,* Cercomonas; note trailing flagellum attached to side of body. Not recognized as a flagellum by some workers. (*Wenyon.*)

trailing flagellum is attached to the side of the body as far as the posterior end, usually being continued as a free flagellum. According to others, this has only a single flagellum the free one being at the anterior end. Bodo and Prowazekia have both two flagella, one waving anteriorly, the other trailing behind. Prowazekia differs from Bodo, and also from Cercomonas, in having a parabasal body. Of these parasites only the Prowazekia is supposed to be truly parasitic in man. The Bodo and Cercomonas are not infrequently found in freshly passed feces, probably occurring as free-living forms which have been ingested accidentally as cysts with water or food. All of these three types are culturable, forming small round cysts.

While the Bodo and the Cercomonas are considered to be innocent forms of infection, in my experience they are capable of producing a dry catarrhal condition of the lower colon and rectum, and while the symptomatology may be negative, local inspection of the

mucous membrane usually shows that some slight pathology exists. Soper has described a dry linear fissure in the membrane, which I also believe is characteristic enough to be diagnostic.

MULTI-FLAGELLATE INTESTINAL PROTOZOA.

Trichomonas Intestinalis.—This is the commonest of the multi-flagellate group. It makes its home in the upper part of the large intestine and cecum, often multiplying in prodigious numbers. It

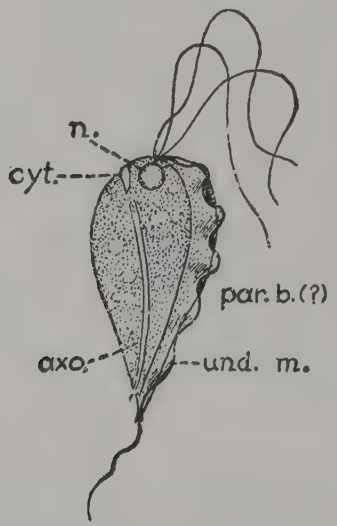


Fig. 112.—Trichomonas intestinalis. *n*, Nucleus. *cyt*, Cytostome. *axo*, axostyle. *par b*, parabasal body (?). *und m*, Undulating membrane. ×2400. (*Wenyon.*)

lives also in the vagina and in the urinary tract, being often found in vaginal discharges, especially in cases of leucorrhea. It has been supposed that the vaginal parasite, which is larger than that of the intestine, is a distinct species, and it has been given the name Trichomonas vaginalis. The probability is that it is identical in form with the intestinal parasite.

The Trichomonas intestinalis is a pear-shaped flagellate averaging about 8 to 15 μ in length, the size being inversely proportional to the rapidity of multiplication. It has three vigorously moving flagella arising from the blunt anterior end and a fourth wavy one which turns backward and is attached to the side of the body by an undulating membrane. Along the line of the attachment of this membrane is a structure which takes a deep stain, known as the chromatic basal

rod and believed by some to be a modified parabasal body. Arising near the anterior end and running through the body is a sort of supporting rod called the axostyle, which is supposed to be used as an organ of locomotion. There is a slight depression or cytostome on the anterior end at one side which serves as a mouth, and a small round nucleus lies in the body just behind the origin of the flagella. Some of the forms have four or five flagella instead of three.

Trichomonas swims by active lashing movements of the free flagella and by wave motions of the undulating membrane. The body revolves as the animal wends its way through the semi-liquid substances in which it lives. Multiplication is by longitudinal division of the body, the flagella and undulating membranes and internal structures all being duplicated before the animal splits in two. Encystment occurs in the Trichomonas by some of the flagellates which escape from the body with the feces, degenerating, and the organism

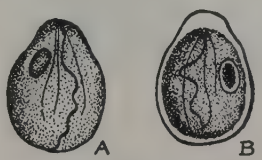


Fig. 113.—Trichomonas intestinalis; *A*, Pre-encystment stage; *B*, Encysted form. ×2000. (*Lynch.*)

taking on a pear shape, although having ameboid movements like the ameba from which it is identified by the undulating movement which persists at one side of the body. Sometimes the cyst takes on an inactive state, becoming active when warm.

The Trichomonas is generally regarded as a harmless parasite, but there is little doubt now that it can be the cause of persistent diarrhea, sometimes very severe and of long duration. Epidemics of diarrhea and mild dysentery symptoms in man, apparently caused by the Trichomonas, have been reported from Peru, Brazil, China, South Carolina and Indiana, and there is no doubt that the parasite is mildly pathogenic wherever it occurs.

No specific drug for use against Trichomonas has yet been found. Methylene blue in weak solutions is absorbed by the parasites and causes them to become round and quiet. Castellani recommends taking methylene blue by mouth and by means of an enema, and with the methylene blue solution the flagellates are said to decrease rapidly and to disappear usually within a few days. Chase and Tasker[7] have drawn attention to the necessity that the methylene blue be of the pure form—namely methylthionin hydrochlorid, and that the ordinary

methylene blue employed in laboratory staining solutions contains traces of zinc. They gave 2 grains (0.12) every three hours by mouth and once or twice a day a high enema which should be retained for fifteen minutes, consisting of 500 cubic centimeters of an aqueous solution of the drug, the strength of which was between 1 to 500 and 1 to 200. They claimed also that the effect of this drug was likewise efficient in the Lamblia.

In the cases that I have treated, comprising the macrostomia and lamblia forms, rapid cure was brought about by a 1 to 500 solution of methylene blue, a liter being given by means of trans-intestinal lavage. After persistent examination of stools there is no doubt that a few

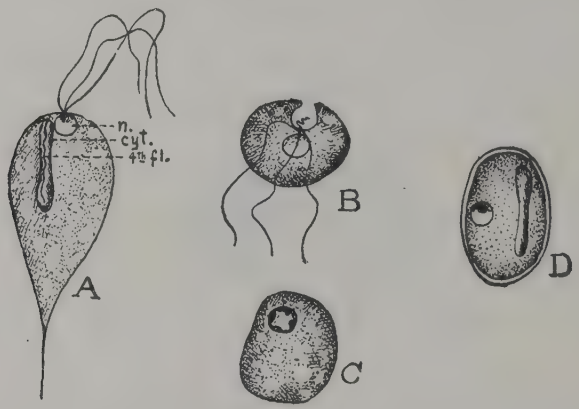


Fig. 114.—Macrostomia (or tetramitus) mesnili. *A*, Adult parasite. *n*, Nucleus. *cyt*, Cytostome. *4th fl*, Fourth flagellum. *B*, End view of adult parasite, showing cytostome with flagellum on it. *C*, Degenerating form, resembling an ameba. *D*, Cyst showing nucleus and cytostome. ×2000. (*Wenyon.*)

of such irrigations brings about a total cure of the condition. Iodine, one grain in a liter of water taken in the evening on three successive days is supposed to bring about a cure of the condition.

The fact that these organisms are probably transmitted through the medium of drinking water and of fresh vegetables which are eaten uncooked after being previously fertilized with human excrement suggests the available methods of prophylaxis.

Macrostomia (or Tetramitus) Mesnili.—This is a parasite which closely resembles the Trichomonas, being somewhat smaller, averaging about 8 to 10 μ in length. It has three slender anterior flagella but no conspicuous undulating membrane. It has a large and conspicuous slit or cytostome along one side which corresponds to the very small mouth cavity of Trichomonas. Within the cytostome is a

fourth inconspicuous flagellum which seems to be attached to a small undulating membrane. The lower end of the organism is drawn out into a long point. This form of flagella may be considered in the same connection as the Trichomonas in symptomatology and in treatment.

Giardia (or Lamblia) Intestinalis.—Next to the Trichomonas this is the most common flagellate in the human digestive tract. Unlike the other forms of the multi-flagellates it establishes itself in the upper part of the small intestine. Its main characteristic is its odd shape, which, according to Wenyon, resembles a pear split into two

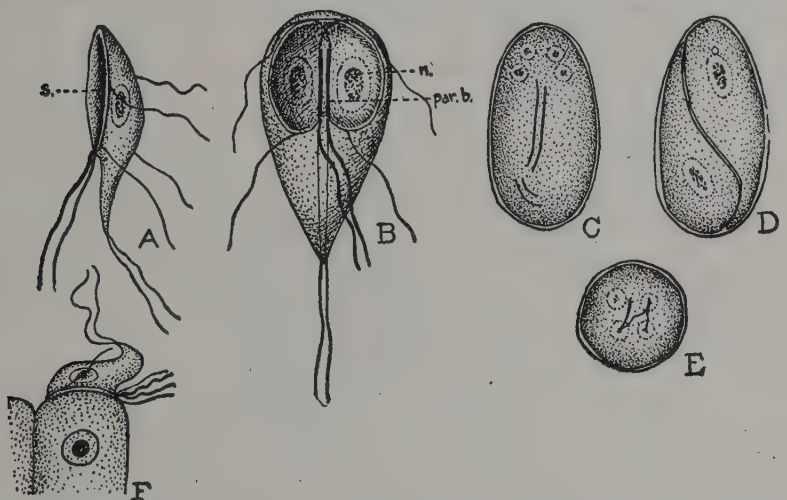


Fig. 115.—Giardia (or Lamblia) intestinalis. *A*, Side view. *s*, Sucker-like depression. *B*, Ventral view. *par b*, Parabasal bodies. *n*, Nucleus. *C*, Young cyst with four nuclei. *D*, Mature cyst containing two parasites. *E*, End view of young cyst. *F*, Parasite resting on epithelial cell. ×2000. (*Wenyon, Grassi and Schewiakoff.*)

parts along the longitudinal axis. There is a flat surface on which there is a sucking disk with raised edge, and a convex surface. The tapering extremity or tail can be turned over the convex back, and it terminates in two flagella. There are three other pairs of flagella, the arrangement of which is best seen by referring to the plate.

The Lamblia intestinalis is remarkable in being bilaterally symmetrical, every organ, including the nucleus, being accurately reproduced on each side of the middle line. The parasite has a comical owl-like appearance, and is 12 to 18 μ in length, and fastens itself to the convex surface of an epithelial cell by means of its sucking disk, resting with its flagella streaming like the barbels of a catfish. It has

been reported that long rows of them have been found resting on the surface of epithelial cells of digestive glands. Miss Porter who has studied this infection in British troops from Gallipoli, estimated that in one case the number of cysts, each having been an active flagellate in the intestine, exceeded 14,000,000,000 in a single stool. The division of this organism takes place after encystment, not much being known about how it occurs otherwise. The mentioned flagellate active parasites become motionless and die soon after leaving the body of the host with the feces, but encysted forms may retain their vitality for a long time.

According to Wenyon this is a very persistent flagellate, often keeping an individual infected for years. It is commonly noticeably pathogenic, causing intermittent diarrhea in which blood and mucus is passed, swarming with parasites. Usually there is an intermittent history to the diarrhea, in the interval of health there being apparently normal stools, when only the cysts may be recovered from them. The infection is common in the East and here lately has been met with by me which makes me feel that it is more or less present in America all the time. Acute symptoms may last from one to six months, after which the symptoms practically disappear for a variable length of time. Usually colder weather, or the patient going to a colder climate, causes a subsidence of the diarrhea.

It is reported by Chandler[8] that Giardia infections are extremely difficult to get rid of, and that some infections seem to survive every attempt at treatment. They do not respond to emetine, and only occasionally to beta-naphthol. Probably the reason for the non-cure of the case is that these little animals have a faculty of lodging themselves in the digestive glands outside the main passage of the intestine, where it is difficult for drugs of any kind to reach them. I am inclined to believe that this is rather a discouraging attitude to take, because the persistent use of methylene blue with beta-naphthol and occasional doses of castor oil seems to be efficient. It must be remembered that this form of multi-flagellate is in the small intestine. It is my belief that the difficulties of treatment have been due to that fact. If the use of the methylene blue solution by trans-intestinal lavage as mentioned above and also by mouth is employed, the result of the treatment is prompt and has been curative in every instance that I have met with.

INTESTINAL FLUKES.

There are several species of flukes which appear to be common parasites in the human intestine in certain parts of the world. They

are most common in the Oriental countries where other forms of human flukes abound the most. Many of these are very small but may occur in great numbers producing practically the same effect as do the tinea, namely anemia, emaciation and general debility. Most often they occur in the feces in individuals in whom there seems to be no special symptoms suggesting their presence.

The smallest fluke known to be parasitic in man is Yokagawa yokagawa, named after a Japanese parasitologist. It is widely distributed in the East and is parasitic in man, mice and dogs. The cysts are numerous in fish which in those countries is eaten raw, and probably that is the way the infection occurs in the intestinal canal. This

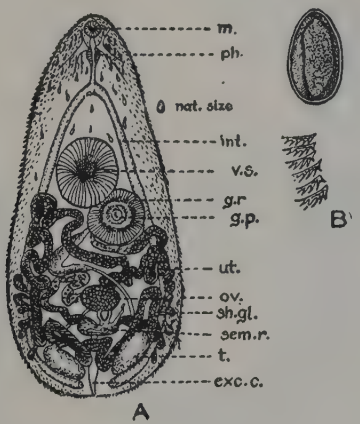


Fig. 116.—Heterophyes. A very small intestinal fluke of man. *A*, Adult. *B*, (×350) Species from genital ring. *g r*, Genital ring. *g p*, Genital pores. *v s*, Ventral sucker. *sh gl*, So-called yolk-glands really shell glands. *ut*, Coiled egg filled uterus. *int*, Intestine. *m*, Mouth in oral sucker. *ph*, Pharynx. *sem r*, Seminal receptacle. *t*, Testes. *exc c*, Excretory canal or ovary, ×33. Egg shown above, ×500. (*Looss.*)

organism inhabits the upper portion of the small intestine, sometimes in considerable numbers, where it can cause enough damage for a persistent type of intestinal catarrh. It is remarkable for the lack of a ventral sucker and is only about 1 millimeter in length and about half as broad. Its body is covered with a great many microscopic spines.

Another form of very similar nature, the Heterophyes heterophyes, which also occasionally parasitizes man. Two species of Echinostoma normally parasitic in other animals occur occasionally in man in the Malay countries. They are distinguished from other flukes by the crown of spines around the mouth sucker.

The Gastrodiscoides hominis is a species which is characterized by the expansion of the posterior end of the body into a great concave disc. It is a small reddish brown parasite a little over one-fourth of an inch in length, and inhabits the cecum and large intestine of hogs, and occasionally of man. It is found mostly in India. A closely allied species occurs in horses and asses in many parts of Africa. This is known as the Watsonius watsoni and is occasionally found in the small intestine of West African negroes.

There are several very large forms of flukes belonging to the genus Fasciolopsis, especially notable being the F. buski, found in many East Asian countries. This species reaches a length of over an

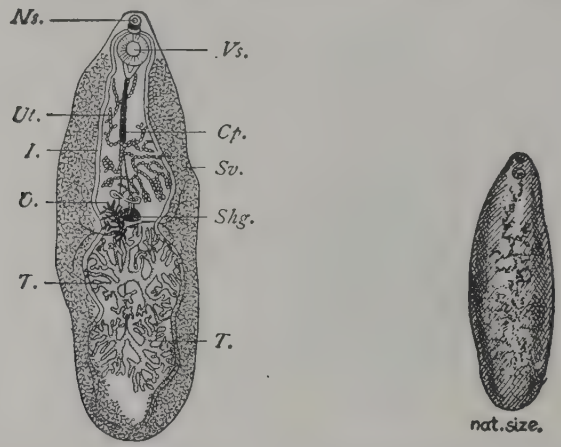


Fig. 117.—Fasciolopsis buski. Large intestinal fluke of man. *Vs,* Ventral sucker. *Cp,* Cirrhous pouch. *I,* Intestinal fork. *Sv,* Vitelline sac. *T,* Testes. *O,* Ovarium. *Ms,* Mouth sucker. *Shg,* Shell-gland. *Ut,* Uterus. (After *Odhner.*)

inch with a width of about half an inch, and has the ventral sucker very close to the mouth. It inhabits the small intestine of the hog but occasionally parasitizes man. The larval stages are said to encyst in shrimps.

The full life history of none of these intestinal parasites is known, and we can only guess at them by analogy with more or less closely related parasites about which we have more knowledge. They do not seem to do much damage other than causing a slight intestinal irritation or catarrh, and sometimes light dysenteric symptoms. They are susceptible to most of the drugs used for expelling tapeworms and round worms. Some species are said not to be affected readily by santonin, though they are expelled by thymol and naphthalene, and presumably by oil of chenopodium.

BILHARZIA.

A very chronic form of dysentery or dysenteric diarrhea associated with bilharzial infection of the large intestine is a very common disease in Egypt. Cases have also been reported from other

Fig. 118.—Schistosomum hematobium (Bilh. 12/1). Male contains the female in the canalis gynecophorus. (After *Looss.*)

parts of the world, particularly in the Panama Canal zone. It is seldom seen as a primary disease, being nearly always secondary to bilharziosis of the urinary organs. However it can occur primarily as an infection of the lower colon and the rectum, the urinary organs being free.

The common site of the disease is the sigmoid flexure and rectum, although the whole of the large intestine may be involved, and the

disease may even spread to the small intestine. The appendix may also be involved. The earliest change is a general congestion of the mucous membrane, in many instances amounting to a general catarrh with small hemorrhages, the mucous membrane having a rosy-tinted color, and velvety in appearance. It is definitely thickened and shows under a hand-lens innumerable tumified, glandular orifices filled with mucus with a layer of extremely tenacious and transparent mucus. This catarrhal congestion is soon followed by the formation of adeno-papillomata, which are at first very delicate and vascular, and may occur singly. They usually appear first near the anus. They soon increase in size and may become complicated in form, and may be sessile or pedunculated. Cases have been described

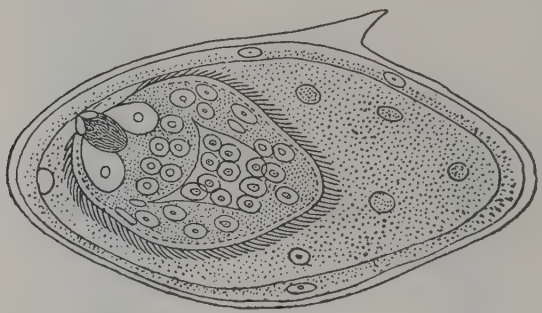


Fig. 118*a*.—Schistosomum hematobium (from feces). (After *C. W. Daniels.*)

in which the masses of these papillomata have filled up the lumen of the gut, and so simulate a new growth which may be palpable through the abdominal wall. In addition to the papillomata, ulceration generally occurs. In many instances the ulcers are formed by the sloughing off of papillomata. Generally at this time a secondary bacterial infection takes place with the establishment of sinuses which may even perforate. Sometimes the lower part of the rectum may collapse. In advanced cases an anchoring of the bowel by a pericolitis, or a periproctitis, interfering with the contractile power and functions of the bowel is characteristic of the disease. This anchoring may be so firm that considerable difficulty is experienced in a post-mortem examination in removing the bowel.

Symptoms.—The disease may be latent and its existence only suspected on the discovery of the ova in the feces, but when symptoms are present they usually are of a very chronic nature. There is a colicky pain in the abdomen, a feeling of fullness in the rectum, the number of motions are increased, and in a bad case, dysentery is

simulated. Pain and tenesmus increase, the pain may be continuous, and prolapse of the rectum often follows. The motions, however, differ from that of chronic amebic dysentery, and even the bacillary form of the disease, in that the quantity of mucus is very large, at times coming away without the passage of stool with it. Sometimes this mucus is slightly blood-stained. Symptoms of urinary bilharziosis may complicate the case, and perhaps even an amebic infection

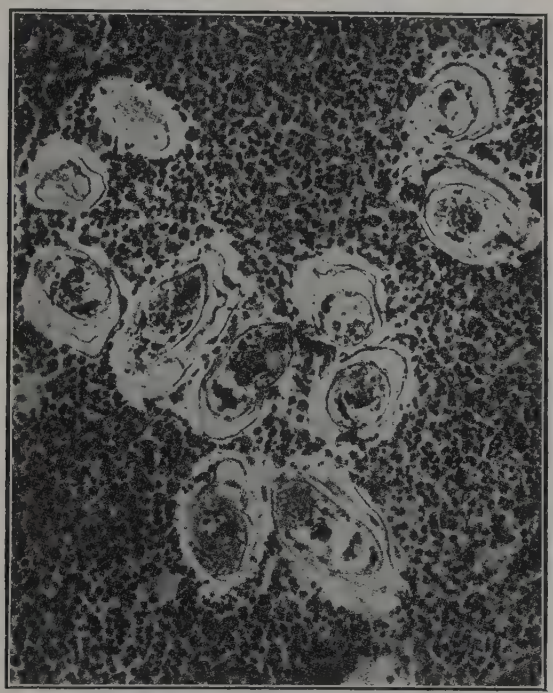


Fig. 119.—Ova seen in the polypoid rectal tissue. (*Meader.*)

may exist. The prognosis is bad as a rule, sepsis and exhaustion closing most of the cases of these complex infections.

At times the case may come under observation with an abdominal tumor, which is usually situated in the region of the descending colon or the sigmoid flexure, although it may occur in any part of the abdomen. This lump generally has existed for some time and slowly increased in size, and may be painful and tender. The disease is that of dysentery with some abdominal pain. These masses in the left side may be nodular and strongly suggest carcinoma. Cases are on record in which the masses have been removed with the suspicion that they were carcinomata, proving on examination to be inflamma-

tory masses of bilharzial adeno-papillomata instead. No criticism could be made of this form of treatment because the incision of the localized growth is undoubtedly the best treatment, as it destroys at the same time the blood-vessels containing apparent worms.

A sigmoidoscopic examination is of very great assistance in confirming the diagnosis, for by its aid the diagnosis is simple. One searches for the existence of papillomata in the lower 10 inches of the bowel, for it must be remembered that amebic ulcers are rarely seen so low, although chronic bacillary ulcers may occur. The presence of papillomata or papillomata and ulcers clinches the diagnosis. The blood examination usually shows eosinophilia although in many chronic cases it is absent.

Treatment.—The treatment is very unsatisfactory, for even were all the living worms to be killed off, the destruction of tissue and the new growths present an almost insuperable obstacle to recovery. It must be remembered, moreover, that the mucosa and the submucosa are crowded with ova, many of which are calcified, and which will continue to irritate for months or years. Accordingly all such drugs as salvarsan, emetine and filix mas are useless. The bowels should be regulated by salines or liquid paraffin. If associated amebic infection exists, emetine should be given. Most of the treatment, however, should be local. Astringent enemata, such as those of tannin or chloride of zinc, and, in case where there is much pain and tenesmus, starch or starch and opium enemata, give relief. But surgical help is often required to remove polypoid growths which irritate the sphincters, or the removal of prolapsed mucous membrane. If definite bilharzial tumors exist which are well localized, they may be removed.

THE TAPEWORMS.

Most remarkable in their structure as compared to other parasites in the intestinal canal are the tapeworms. A mature tapeworm is not an individual, but a whole family, consisting sometimes of many hundreds of individuals one behind the other like the links of a chain. What is peculiar about them is that they have no digestive tract, but by their long length being constantly bathed in the fluids of the digestive tract they absorb nutriment for their nutrition.

In general form they are long tapelike organisms which attach themselves to their host's intestinal walls by a "head" or scolex at what is really the posterior end of the chain of segments. This scolex is furnished with suckers and often hooks. Next to the head is a narrow region or neck, which continually grows and forms segments,

each new one produced pushing forward the segments previously formed. Thus is produced the characteristic chain of segments, each of which is known as a proglottid (segment). The oldest one of these is at the end of the chain, those just back of the neck being young and immature. There is a long brain-like mass running from the

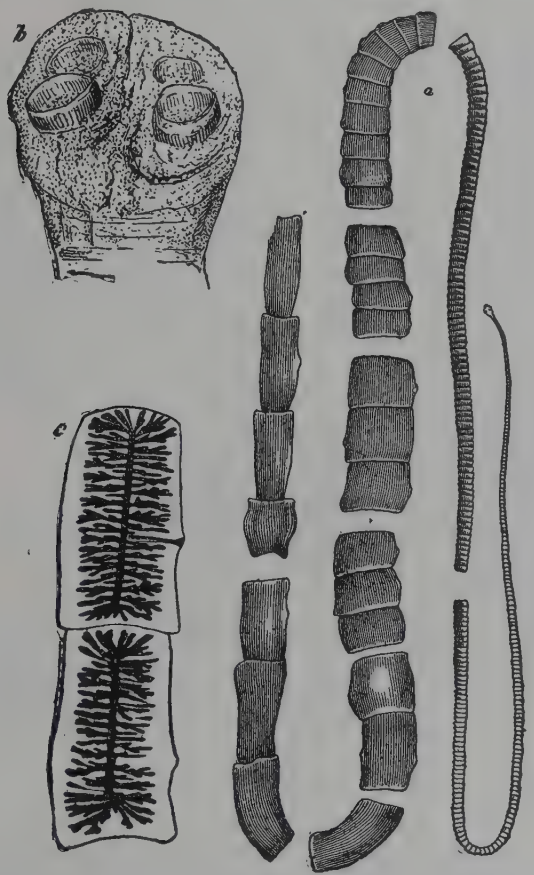


Fig. 120.—Tenia saginata. *a*, Natural size of the worm at different sections. *b*, Head (with pigment canaliculi). *c*, Proglottides. (Partly after *Leuckart*.)

scolex in two longitudinal nerves which continues through all the proglottids in the chain. The muscles and excretory canals also run continuously through the chain. Each proglottid, however, possesses reproductive systems of its own, these being bisexual. The female system consists of an ovary, a pair of shell glands, a seminal receptacle for receiving and holding the sperms until used for fertilization, a vagina for the entrance of the sperms, and a uterus for the storage

of the mature fertilized eggs. The male system consists of a number of scattered testes for production of sperms, all connecting by minute tubes with the sperm duct. The latter, near where it opens at the surface of the body, enlarges into a pouch where the mature sperms are temporarily stored. The sperm duct ends in an extensible copulatory organ for conducting the sperms into the vagina of the same or another proglottid. Copulation takes place by the doubling back of the chain of proglottids on itself bringing the young mature male segments into contact with the older mature female segments. After copulation when the mature fertilized eggs begin to form,

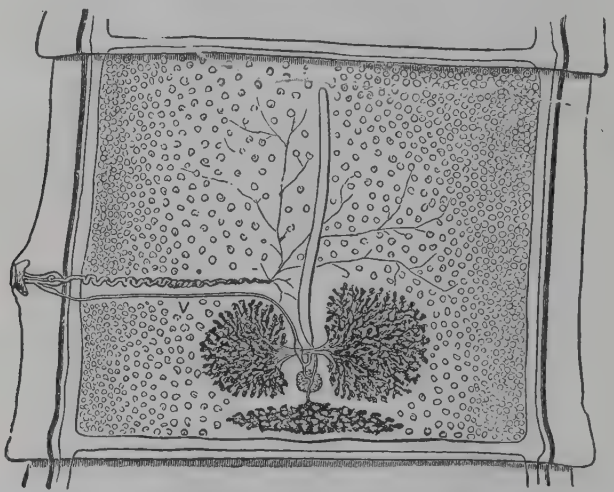


Fig. 120*a*.—Proglottid of Tenia saginata, with genital organs. Note that the vagina, *V*, passes round the left ovary and not through it. (After *C. W. Daniels.*)

changes take place in the proglottid. The uterus enlarges and branches until it nearly fills the segment, crowding aside and absorbing the other organs. When the segments are thus distended with eggs they are spoken of as ripe proglottids and are ready to break loose from the chain to be voided with the feces of the host.

The life histories of all the tapeworms are much alike. Usually before the ripe proglottids become detached and pass out, the eggs develop inside their tough shell into little round embryos with six hooks. In order to continue their development such embryos must be eaten by another species of animal which acts as an intermediate host. The adult form of the worm occurs in carnivorous animals, while the intermediate host in which the larva develops is a herbivorous animal, but there are exceptions to this. When eaten by a suit-

able host, the shell enclosing the six-hooked embryo is dissolved off, and the embryo is released. It soon migrates into the organs and tissues of the body, generally in the muscles in which it awaits its chance for further development which only arises when the animal is eaten raw by another. The meat of such an intermediate host

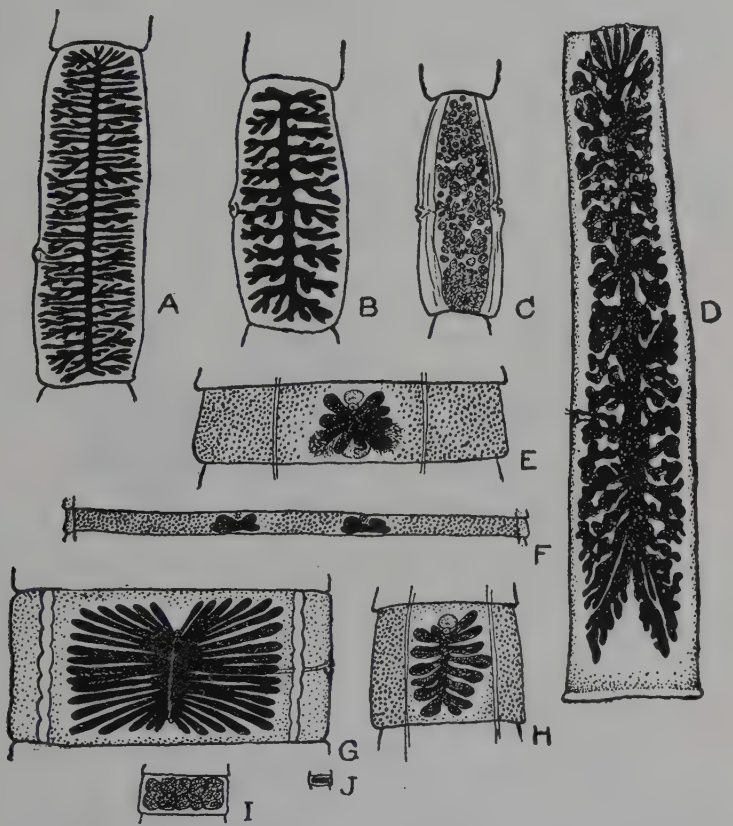


Fig. 121.—Ripe proglottids of various tapeworms of man, drawn to scale according to average measurements. *A*, Tenia saginata. *B*, Tenia solium. *C*, Dipylidium caninum. *D*, Tenia confusa. *E*, Dibothriocephalus latus. *F*, Diplogonoporus grandis. *G*, Dibothriocephalus cordatus. *H*, Tenia Africana. *I*, Hymenolepis diminuta. *J*, Hymenolepis nana. (After *Leuckart, et al.*)

thus affected, represented in cattle, sheep, hogs, fish, etc. is known as "measly" meat.

When the tapeworm is in a suitable intermediate host the embryo loses its hooks and grows into some form of bladderworm. That is, the body undergoes a series of transformation which usually results in the formation of a bladder-like body filled with a watery fluid. In

this form the head, or scolex is developed. The larvæ of the Dibothriocephalide group are quite unlike the bladderworms of other tapeworms. They grow as long wrinkled wormlike bodies with the head invaginated in a little projection at the anterior end.

When the organs or tissues in which the larval stages of tapeworms are developed are eaten by an animal of a kind from which the eggs originally came, all but the head of the bladderworm is digested off. The head then attaches itself to the wall of the small intestine with the aid of its sucker and hooks, and begins to bud off proglottids of another generation.

Symptoms.—Although tapeworms do seem to exist in some people without the production of symptoms, in the majority they produce distinct effects in the impairment of nutrition. It is very

Fig. 122.—Types of tapeworm larvæ. *A*, Cysticercus of Tenia solium with head and neck evaginated ×3. *B*, Cysticercoid of Hymenolepis nana ×12. *C*, Plerocercoid of Dibothriocephalus latus with head invaginated. (*Braun-Chandler.*)

probable that in addition to these, the symptoms produced are due to resorption of toxic material from the worms. They act distinctly in the way of interfering with normal peristalsis either in a mechanical way or because of a toxic effect of their excretion upon the neuromuscular apparatus of the gut. The amount of food taken from the host for nourishment by a growing embryo produces a ravenous appetite. I have met with several instances of chronic diarrhea produced by tapeworm due to the toxic effect of the worm, or in some way the worm encysts by causing destruction of the mucous membrane and ulceration. In depriving the host of nutriment, sometimes an extreme anemic condition ensues in which the individual may be weak and considerably exhausted. This may be shown in his appearance, and in not a few cases, there is considerable mental disturbance.

Abdominal pain, anal itching, disordered appetite and digestion, emaciation, anemia and many types of nervous derangements, as gid-

diness, partial paralysis, false sensations and epileptic fits, are common symptoms of tapeworm infection. There is little doubt now that the nervous symptoms are due to the intoxicating substances liberated by the worms.

Diagnosis.—Diagnosis is usually accomplished by the patient describing and bringing in segments of the worm which may be voided without stool. A search of the stool for the ova, or the presence of a marked eosinophilia, and perhaps the description of symptoms mentioned above suffices for diagnosis.

Treatment.—The diagnosis being made, a preparatory treatment is usually called for, and this consists of putting the patient on a light diet and the bowels being moved with castor oil or calomel so that the parasite can meet with no obstruction in its passage through the intestine. The drugs which have been found most useful in expelling tapeworms are male fern, pelletririne, and thymol. These drugs are dangerous if not taken properly, and none should be taken without the supervision of a physician. Thymol, for instance, while ordinarily harmless, since it is not absorbed by the intestine, is soluble in alcohol and certain other substances and may cause death if taken along with these things. Oil of chenopodium, which has recently come into great favor for expelling hookworms and is even more efficient for certain other mematodes, has been found valuable for expelling dwarf tapeworms, and would probably be equally effective for other species.

After the drug has been taken, a purgative is given for the purpose of driving the parasite out. It should be passed into a vessel of warm water, since sudden contact with cold stimulates the nervous system of the worm and causes it to contract suddenly, thus often breaking it up before it has been completely expelled. A careful search for the head should be made, and if not found the treatment should be repeated in the course of a week or ten days. In my experience with most of the common tapeworms when the head has not been removed, it takes about nine weeks for segments to reappear in the stool. If the head has not been found, and the symptoms which the patient suffers from are few, three or four months may be allowed to go by, and if the segments do not reappear, the probabilities are that the head of the worm has been passed.

For a long time it has been my custom to employ the detail of treatment suggested by Fowler,[9] the method of which is as follows:

The patient is kept in bed.

For two or three, or in some cases four, days the patient is given a diet consisting of beef tea, 2 pints; two rusks. During the same period the patient takes tablets of cascara sagrada (gr. ij.) three

times daily. On the fourth day (usually) at 5 A.M, senna; at 9 A.M., at 9.15, at 9.30, and at 9.45 a capsule containing 15 minims of the extract of male fern; and at 11 A.M., senna. If by 1 P,M. the worm has not been passed and the head found, a second course of treatment with male fern at intervals of fifteen minutes is begun; to be followed in an hour by a purgative draught. If the head is not found, a third course of treatment is prescribed.

It is rarely advisable to continue the treatment beyond this without an interval of a day, as the patient may be somewhat exhausted.

In some cases the head is not found until the treatment has been repeated four times, and in still others the treatment has to be repeated six times.

Having regard to the prolonged duration of the affection in many of the cases and to the frequent failures of previous attempts to dislodge the parasite, it may be fairly claimed for this method that it presents advantages over that which is apparently still generally adopted.

The chief points of difference are:

1. Complete rest in bed. It is not advisable for patients taking such an inadequate diet as is necessary, to continue at their ordinary occupation.

2. Prolonged period of restricted diet. The usual instructions given to out-patients were to eat no solid food after midday and to take the draught prescribed on the following morning; this to be followed by a purgative an hour later. The following, which is taken from a well-known text-book of therapeutics, shows that this method is still approved.

The oil (male fern) should be given in the morning on an empty stomach, the bowels having previously been evacuated. At noon an ordinary meal may be taken, and in the evening a brisk purge.

To insure that the bowels shall be as nearly empty as possible when the vermicide is administered, it is necessary that a diet which leaves little solid residue shall have been taken for some days, and that for the same period a laxative such as cascara sagrada, which is said to act most efficaciously when given in small and repeated doses, should be used. The duration of the period of restricted diet depends upon the strength of the patient.

3. The method of administering the vermicide. If a single dose is given, it is conceivable that it may pass rapidly over the worm and fail to destroy it, whereas it is possible that the prolonged action of repeated doses is more effective. The nauseous taste of the drug is avoided by the use of capsules.

4. The search for the head of the worm. As the worm usually breaks at a distance of about 1½ inches from the head, and as the portion left is an exceedingly slender filament, a very careful search is necessary to find it. To facilitate this, the pan into which the motion is passed should be covered with black crape, as against this the fine white filament terminating in the head is more readily seen.

It was observed that the head of the worm was found in 17 out of 22 cases, and that a cure was certainly effected in 2 out of the remaining 5 cases, 86 per cent. in all.

There appears to be a belief that oil of male-fern is less successful against Tenia mediocanellata than against Tenia solium and the Bothriocephalus latus. This series of cases, so far as it has any bearing on the point, does not support such a view. None of the very various toxic symptoms described as having occasionally followed the administration of this drug were observed.

In clinic work I have had good success with an initial dose of castor oil, the taking of nothing but black coffee for one or two days, and then giving the following prescription, one-half being taken in the evening and one-half in the morning.

℞	Cortic granat.	100.0
	Sem. Pepon.	15.0
	Ext. filie Alth.	4.0
	Pulv. Ergot	0.65
	Pulv. Acac.	6.0
	Ol. Croton	0.065
	Aquæ, Ad.	250.0

Fiat Mixt.

Sig.: Take one half bottle at bedtime, the remainder in the morning.

I believe, however, that the bed treatment is by far the better method; the percentage of worms recovered by that method is greater than the ambulatory one. Both thymol and oil of chenopodium have been used by me with success in cases that resisted the above method of treatment, and as time goes on I am more inclined to the efficiency of oil of chenopodium as a vermifuge at least equal to, if not better, than that of male fern.

Prevention.—Prevention, of course, varies with the species of tapeworm and its intermediate host, but since infection with all the common human species, with the exception of the Hymenolepis, occurs from eating raw or imperfectly cooked meat of some kind in which the bladderworms have developed, the exclusive use of thoroughly cooked meat is the best preventive measure. Experiments show that pork bladderworms are killed when heated to 127° F.

and beef bladderworms to 120° or even less, but the difficulty of heating the center of a large piece of meat even to this point is shown by the fact that in an experiment to test the penetration of heat, a ham cooked by boiling for two hours had reached a temperature of only 115° in the center. Beef which has lost its red or rare color is quite safe.

The bladderworms in meat are usually destroyed by the ordinary cold storage, within three weeks in the case of the beef bladderworm, but not always so soon in the case of the pork bladderworm. Thorough curing or salting of meat is also destructive to the parasites.

Infected persons should be careful not to contaminate the food or water of domestic animals with their feces, bearing in mind the various ways in which the eggs may be disseminated.

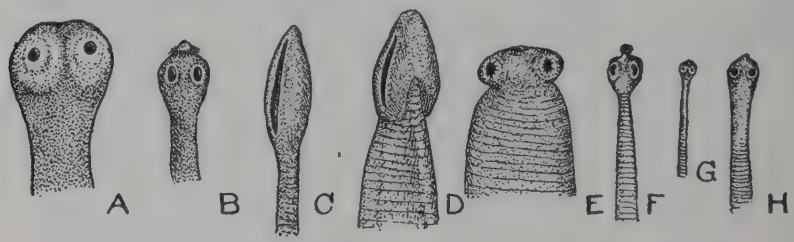


Fig. 123.—Heads of some adult tapeworms found in man. *A*, Beef tapeworm. *B*, Pork tapeworm. *C*, Fish tapeworm. *D*, Heart headed tapeworm. *E*, African tapeworm. *F*, Double-pored dog tapeworm. *G*, Dwarf tapeworm. *H*, rat tapeworm ×10. (*Chandler.*)

The tapeworms of man belong to two quite distinct families, the Teniide in which the scolex is rounded and furnished with four cup-shaped suckers, and the Dibothriocephalide, in which the head is flat and possesses two slit-like suckers. The latter family differs from the Teniide in having eggs with lids like those of the flukes, and without developed embryos when passed in the feces.

FAMILY TENIIDE.

Beef Tapeworm.—The commonest tapeworm in the human being found throughout the world is the beef tapeworm, Tenia saginata. The adult of this species as it occurs in the human small intestine consists of over 1000 proglottids, and grows to a length of 15 to 20 feet. Specimens as long as 40 feet have been reported. More than one worm may be present in the human host at the same time.

The scolex of the beef tapeworm is hardly larger than the head of a pin. It possesses four small suckers for adhering to the wall of

the intestine, but there is no crown of hooks. The suckers are apparently quite sufficient for maintaining a hold.

The proglottids gradually increase in size as they get farther from the scolex, and the organs contained in them developing. The general form of a sexually mature proglottid and the appearance and arrangements of the organs are shown in the accompanying figure. Shortly after sexual maturity has been reached and the sperms for fertilizing the eggs have been received, the uterus begins to grow and develop lateral branches to accommodate the rapidly forming eggs.

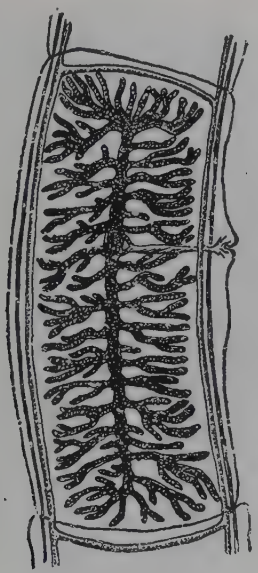


Fig. 124.—Gravid segment of beef tapeworm; Tenia saginata ×4. (*Stiles.*)

The proglottid then enlarges, resembling a pumpkin seed in shape. At this time the greatly developed uterus, distended with eggs, occupies practically the whole segment, while nearly all the other organs degenerate.

A man infested by a beef tapeworm expels several hundred proglottids a month, each one gorged with thousands of eggs. These are disseminated by rain water, washed by streams into drinking troughs, carried about on the feet of flies, adhering to the heel of a boot, and in many other ways the eggs passed with the feces may be transferred to the grass or water eaten by cattle. When they are taken by the cattle the six-hooked embryos escape from the eggs and migrate into the muscles of the new host, attacking especially the muscles of mastication. Here in the course of from three to six

weeks they grow into bladderworms, which are grayish in color with a little yellow spot where the head is invaginated. Beef which contains bladderworms is said to be "measly." Infection of man results from eating measly beef which is raw or only partially cooked.

Pork Tapeworm.—Common in some parts of the world, but not as rare in the United States as was assumed to be is the species Tenia solium, which passes its bladderworm stage in hogs. When such measly meat is eaten infection with this tapeworm is likely to occur.

Fig. 125.—Photomicrograph of the fore end of the head of the Tenia solium. Center showing the costellum with its double row of hooklets. Edges, the four pigmented suckers.

The adult Tenia solium differs from the beef tapeworm chiefly in the form of the scolex which in addition to the four suckers is armed with a double row of hooks, arranged on a conical projection or "rostellum" at its apex. These worms usually are of less length than the beef tapeworms, seldom exceeding from six to ten feet, containing about 800 to 900 segments. The ripe proglottids are of smaller size and have fewer branches of the uterus than the beefworm.

The eggs, passed in the ripe proglottids with the feces, develop into bladderworms when eaten by hogs or certain other animals. This is encouraged in the hogs in the way the hogs are housed and fed, and on many farms the hogs are allowed to run loose particularly

the young ones, where they come in contact with privies, their meat then becoming measly from eating tapeworm eggs.

As soon as the eggs reach the intestinal canal, the six-hooked embryos are liberated from the enclosing capsule and make their way through the wall of the intestine, to be carried by blood-vessels to places where they are to develop, which is in almost any or all of the organs or muscles of the hog's body, but they especially favor the tongue, neck and shoulder muscles, and, next in order, certain muscles of the hams, where they may be as numerous as several thousand bladderworms to a pound. These bladderworms are seen from one-fourth to three-fourths to one inch in length.

Unlike the beef tapeworm, Tenia solium can pass its bladder-worm stage in a number of animals, namely hogs, man and dogs. The fact that the larval stage can develop in man makes the species

Fig. 126.—Egg of the dwarf tapeworm (Hymenolepis nana) of man, greatly magnified. (After *B. H. Ransom.*)

particularly dangerous on account of the possibility of self-infection, either by contaminated hands or by a reversal of the peristaltic movements of the intestines which throws the ripe proglottids of the worms back into the stomach where the embryos in the eggs are liberated by the gastric juice. For this reason the Tenia solium is a much more dangerous possibility than infection with other tapeworms.

The Dwarf Tapeworm.—The dwarf tapeworm, Hymenolepis nana is the smallest tapeworm found in man, but it often occurs in such numbers as to cause much irritation in the intestine. It is found oftenest in Italy, the warm parts of Europe, Asia, Africa and America. In the United States it is more common than it has been supposed, and it is easily overlooked unless the feces are microscopically examined for eggs. It is probable that rats and mice are as good hosts for this form of worms as is man.

The adult worm, which consists of from 100 to 200 proglottids, is usually a little over an inch in length and less than one millimeter in width. The scolex has four tiny suckers and a crown of little hooks. The ripe proglottids differ from those of the large tape-

worms in being much wider than long, with an enlarged uterus in the form of a solid mass, partially divided into compartments instead of being branched.

Stiles[10] in 1903 predicted that further investigations would show that Tenia nana is one of the commonest intestinal parasites in the United States.

This worm is commonly believed to pass both its larval and adult stage in a single host, contrary to what occurs, so far as is known, in any other tapeworm. It is said that the eggs of this parasite can be found in the feces within a month after an egg from the preceding generation has been swallowed. Infection with these eggs rarely occurs since the eggs will not develop unless acted upon by the gastric juice. The common presence of this parasite or a variety of it in rats

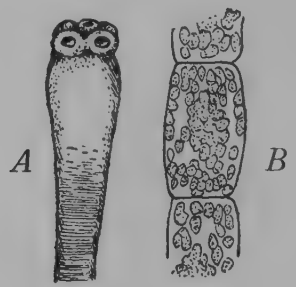


Fig. 127.—Davainea Madagascariensis. *A*, head and neck. *B*, Gravid proglottids ×8. (*A*, after *Blanchard; B*, after *Daniels.*)

and mice indicates that infection in man may occur from accidentally swallowing the excretion from these animals. A single small roll of the black feces may contain as much as one hundred eggs.

Among some of the peculiar symptoms which have been mentioned in connection with this infection is the possibility of a facial edema like that seen in the primary facial edema in trichonosis. Disturbances of the nervous system are marked, particularly that of the sympathetic nerve. The worm buries itself in the intestinal mucosa in which its hold is usually quite deep by the twenty-four or thirty hooklets it has. Chronic spasms and epileptiform convulsions have been reported.[11] The fact that some cases have lasted as long as three years in spite of several apparently successful courses of treatment, seems to point to an infection as the main feature of the dwarf-worm carriers.

A closely allied species, Hymenolepis diminuta, occurs rarely in man. It closely resembles the dwarf tapeworm but is of larger size (4 to 24 inches in length) and has no hooks on the scolex. The eggs

develop in the larva or adult of the mealworm, and in adult beetles. These, eaten by rats, mice or men, are transformed into adults. The eggs have been found in the feces of man fifteen days after the eating of an infected mealworm. It is evident that prevention consists in guarding carefully against the accidental swallowing of mealworms with cereals or other foods, and in cautioning children against putting beetles or other insects into their mouths. This worm in man is rare, though it is common in rats and mice.

Teniide.—A considerable number of other tapeworms of this family have been found in man, accidentally occurring in him, or

Fig. 128.—A way in which tapeworms and other infections may be spread. (From Riley and Johansen's Medical Entomology.)

having a very limited distribution. Of these should be mentioned two species of Davainea. One, D. madagascariensis, which is a small tapeworm reaching the length of ten or twelve inches. It is is found chiefly in children in tropical countries, especially in islands and seaports and on ships. It has been suggested that the intermediate host is the ubiquitous sea-going cockroach. This tapeworm is interesting in that there is not only a crown of hooks on the head, but there are hooks on the suckers also. The other species of the Davainea is D. formosana, recently described from children in Formosa and Tokio. It differs from the preceding species in its larger size, lack of hooks on the suckers, larger size of egg masses in the ripe proglottids and other minor details.

The African tapeworm, Tenia africana, is a species found in German East Africa. It is said to be about four feet in length with

no hooks on the scolex, and with an unusual fanlike arrangement of the uterus in the ripe proglottids. It has been suggested that the zebu may be its host since its flesh is eaten raw by the natives.

A medium-sized tapeworm, Tenia phillippina, reaching a length of about three feet has been found among prisoners at Manila. It closely resembles the African tapeworm. Other species of this have been described from various parts of the world, especially southern Asiatic Russia.

Another form of tapeworm is the Tinea confusa which has been described by Ward in Nebraska. It evidently is very rare.

Of the accidental tapeworms of man there should be mentioned especially the dog tapeworm, Dipylidium caninum. This species is abundant in dogs, and sometimes cats, in all parts of the world. It is a species about a foot in length, with three or four rows of hooks on the rostellym, and a double set of reproductive organs in each proglottid. The larva occurs in lice and fleas. The eggs hatch out in the intestine of the flea larva, and the embryos pass to the body cavity. Children who play with dogs are occasionally infected by this worm probably by accidentally swallowing lice or fleas or by crushing them and then putting the infected fingers into the mouth.

FAMILY DIBOTHRIOCEPHALIDE.

The tapeworms of this family are characterized by a flattened head with two slitlike suckers. The larvæ, which usually develop in fishes, have a long wormlike body with an invaginated head at one end.

Fish Tapeworm.—The common fish tapeworm of man, Dibothriocephalus latus, is an important species in the districts in which it occurs. It is found in all countries where fresh-water fish is extensively eaten, and especially in countries where it is commonly eaten raw. Fish taken from the Great Lakes frequently have been found infected.

The fish tapeworm is a large species and commonly reaches a length of from 6 to 30 feet, or even more, having up to as high as 4000 short, broad proglottids, only the terminal ones of which are as long as broad. Unlike the tapeworms of the family Teniide, the genital openings are near the middle of the under surface of the proglottids, instead of at one side. The proglottids do not retain the eggs until they break off from the chain, but void them, as do flukes, through the genital pore.

The eggs are large and brown with a lid at one end as in fluke eggs, containing six-hooked embryos which are furnished with a covering of cilia. The eggs hatch in water after several weeks and the

embryos swim for a time by means of their cilia, though they often slip out of their ciliated envelope and creep on the bottom. It is believed that the embryos first enter some small aquatic animal, probably a crustacean, which is eaten by a pike or perch or some other carnivorous fish, in the muscles of which it develops. When eaten by a susceptible host in raw or imperfectly cooked fish, the larva, except the head, is digested, and the head, attaching itself to the wall of the small intestine, begins to grow into an adult worm at the rate of about 31 to 32 proglottids a day. The mature eggs begin to appear in the feces in about a month.

This tapeworm is important in the production of intense anemia, probably due to the oleic acid contained in its head which is

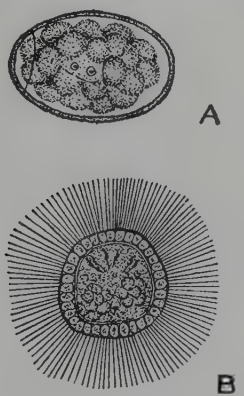


Fig. 129.—An egg of fish tapeworm D. latus. *A*, Segmented condeleis and operculum. *B*, Ciliated embryo of same, × about 300. (After *Looss.*)

a substance having blood-destroying properties. Usually there is a marked effect upon the nervous system in the presence of infection by this type of worm.

LARVAL TAPEWORMS IN MAN.

The most common of these and the best known is the Echinococcus hydatids. It is a miniature form of tapeworm found in dogs and sometimes in other carnivorous animals. It measures only from one-tenth to one-fifth of an inch in length. The human infection usually results from too intimate association with dogs, and children especially are liable to infection by allowing dogs to kiss them or lick their faces with a tongue, which in view of the unclean habits of dogs, may be an efficient means of transmission for the tapeworm eggs.

The hydatids develop in many parts of the body, the liver being the favorite site, the lungs, kidneys, spleen, intestinal walls, mem-

branes lining the body cavity, heart, brain and various muscles may be affected. The development is very slow, it taking about five months for an embryo to reach the size of a walnut. A characteristic feature is that the membrane of the bladderworm itself is very delicate, but the capsule formed by the host is thick and tough. Since this form of worm produces mostly symptoms in the liver, and practically none in the intestinal canal, it need not be described further.

Other forms of the larval worm are the Cysticercus (the Tinea solium which has been described), and the Sparganum mansoni which is a long, elastic rubber-like worm, varying from about 3 to 14 inches in length. This type of worm affects the connective tissues generally under the skin, and need not be described further.

HOOKWORM.

For many years it was customary in the United States to look upon the shiftless people to be found in our South as the product of wanton laziness and an inborn lack of ambition. It is now known that the "poor whites" of the South whom the Northerners considered as "good for nothing" and "irresponsible" were largely instances of hookworm infection. Principally through the investigations of Dr. C. W. Stiles, of the U. S. Public Health Service it was found that these hopelessly incapable and pitifully emaciated and stunted people were the victims of this infection, which sapping their vitality and poisoning the system, stunted both mental and physical growth and that at least over two million people in our southern states were victims of these parasites.

What is interesting in this connection is that the hookworm was discovered in Italy over 75 years ago, and it was only recently that it has been found out that people in most every warm country in the world have more or less instances of infection of this type, in some places at least as much as 100 per cent. being the victims. It has been stated that it is well within the truth to say that over half a billion people in the world are infected with hookworm, it being found more or less prevalent in the entire zone lying within 30° north and 30° south of the equator, and practically all of the tropical and semi-tropical countries. In 1904 more than 300,000 persons were treated for hookworm disease in Porto Rico, and in the United States the disease is found throughout the states south of the Potomac and Ohio Rivers, in Arkansas, Missouri, Oklahoma and Texas, and also in California. Generally speaking, the heaviest infection is found on the light sandy soil of the coastal plains and the lightest infection on the stiff, clay

soil of the Piedmont region, an intermediate infection being among the foothills and mountains. It is peculiarly a disease of the agricultural districts, and in eleven of the southern states between 1910 and 1913, 43 per cent. of the children were found infected, and in the same territory, 35 per cent. of all.

It is therefore remarkable that an infection that was discovered as long ago as this one was, took so long for its significance and widespread areas of activity in the human being to be noticed. Thus hookworm disease has been insidiously spreading, unrecognized and unchecked in the countries of the globe having a mild climate. The infection is in most instances so insidiously acquired by the unsuspecting victim, that he and the members of his family who are probably likewise being affected, do not know just when the disease began to manifest itself. In the course of a few summers, however, a once healthy family has become pale and puny, a once industrious family has become languid and backward in its work, a once prosperous family has fallen into debt, a once proud family owning valuable property has been reduced by an easily curable and easily preventable disease to tenancy and to poverty.

Major Frick reporting on the infected troops at Camp Beauregard from March 1st to September 1st, 1918 showed that hookworm infection is still present in a large percentage of the young men of the South. In many of these, it seemed to have no marked effect upon the health this being due probably to the mildness of the infection or the physical condition of the men through training.

In America the hookworm, Necator americanus, was probably introduced from Africa by the slaves. In most other warm parts of the world, a closely allied species, the Old World hookworm, Ancylostoma duodenale, is more prevalent. It is impossible now to know what was the origin or natural distribution of either species, since both worms have been introduced by infected travelers into every quarter of the globe. In Europe, Ancylostoma duodenale is far the most common.

The two species of human hookworms are similar in nature; they agree in all important details of life history and both produce the same symptoms, require the same treatment, and can be prevented in the same ways. They are round worms, belonging to the great group of nematodes, which in adults live in the small intestine of their hosts and suck blood. The American hookworm is smaller than the Old World species, the measurements being about 8 millimeters and 10 millimeters respectively in the males, and 10 millimeters and 15 millimeters respectively in the females. They are normally whitish in

color but when gorged with blood they are reddish brown. The females are much more numerous than the males, having simple cylindrical bodies, largely occupied by the threadlike ovaries and

Fig. 130.—Necator (uncinaria) americanus hookworm, Porto Rico ×16.4.

egg-filled oviducts. In the Old World species the mouth is armed with a number of chitinous hooklike teeth, which in the American species are replaced by hard ridges or lips. The male worms are also

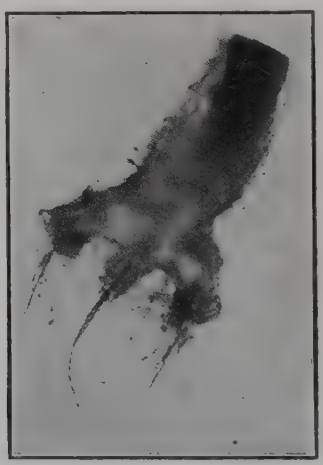


Fig. 131.—Necator (uncinaria) americanus hookworm, bursa of the male ×61.6.

cylindrical but instead of tapering at the tail end they possess an umbrella-like expansion known as a bursa, which is supported by claw-like rays somewhat suggestive of the ribs of an umbrella. The bursa is

used for holding the female during copulation. It was the claw-like ribs of this umbrella which first suggested the name "hookworm" for the parasites, although the hooklike teeth in the mouth of the Old World species might just as readily have suggested the name.

The female worms produce an enormous number of eggs which are poured into the intestine of the host, usually in a continuous

Fig. 132.—American hookworm, showing manner of attachment to intestinal wall. (*Ashford and Igaravidez* from *Gray.*)

stream, but occasionally with intermissions, to be passed with the feces. The thin-shelled eggs, which are about 60 μ by 35 μ in size, and slightly larger in the American species, undergo the first stages of development while still in the intestinal canal, and by the time they are voided with the feces they are segmented into from two to eight

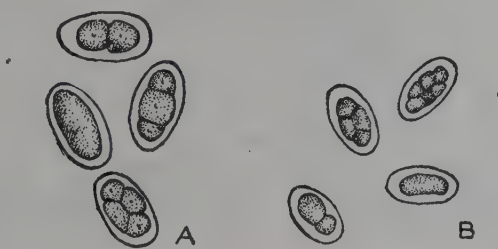


Fig. 133.—Eggs of hookworm in early stages of segmentation. *A,* Necator americanus. *B,* Ancylostoma duodenale. (*Chandler.*)

cells. The segmented condition, together with the fact that they are clear and not yellow or brown from bile stain, distinguishes the eggs from those of many other worms found in the intestine. Further development does not take place until the feces are exposed to air, when, if moisture is present and the temperature is moderately high, the development continues and the embryo hatches in from 24 to 48 hours. The newly hatched worm is about 0.2 millimeter in length with

a bottle-shaped esophagus, a simple intestine, and practically no reproductive organs. According to Looss, the larvæ will not develop well in feces derived from a purely vegetable diet, a small proportion of animal matter being essential for food. If suitable conditions are present the larvæ grow rapidly for four or five days, shedding the skin at the end of the second day, and in about five days under ideal

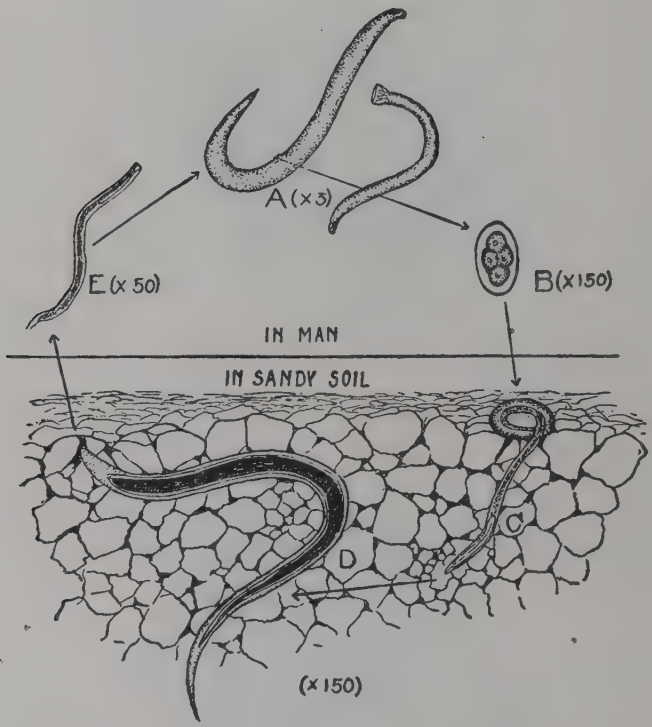


Fig. 134.—Life history of hookworm. *A*, Adults—females and males, in intestine. *B*, Egg passed in feces. *C*, Embryo hatching on ground 24 to 48 hours later. *D*, Fully developed larva enclosed in sheath, ready to infect human being. *E*, Larvæ released from sheath migrating in body of new host. (*Chandler.*)

conditions, the skin begins to become detached again but is not shed. This skin is retained as a protecting sheath for the larva, but does not hinder free motion. The larva by this time has grown considerably in size, being over 5 millimeters in length, and is now in its infective stage and ready to begin its parasitic life. No further food is taken but the parasite begins an active migration in the neighboring soil or water. The parasite is capable of living for perhaps ten or twelve months under favorable conditions of warmth and moisture. They cannot

develop beyond this point unless they gain entrance into the body of the human being and find their way into the intestinal canal, usually the upper portion of the small intestine. The glowing heat of the summer and the freezing weather of winter are destructive both to the

Fig. 135.—*A*, Girl (aged 16), practically an invalid from childhood, has been treated for malaria and tuberculosis. Was found to be heavily infected with hookworms and was given treatment. *B*, As she is today. (*Farrell.*)

development of the hookworm eggs and to the life of the larva. These larvæ may attach themselves to food, carried in on soiled hands, or perhaps more frequently, may be swallowed along with uncooked food such as strawberries, plums, celery and lettuce.

Though some infection is carried through the mouth, most of it is acquired through the skin in a very interesting way. In brief, when

they are protected by the second sheath, they possess the faculty to burrow in a few minutes through the skin and to enter the tiny blood capillaries, then being carried along by the blood current to the heart and the lungs. After they are carried into the lungs, the capillaries being too small to permit them to pass through, they begin to burrow again and get into the air-spaces and finally into the windpipe and then the throat, where they are swallowed with saliva and food. Usually in about eight weeks from the time of their entrance through the skin, the host is harboring adult hookworms in the intestinal canal. Once in the intestinal canal, a moulting takes place and the development into the adult worm, which can be seen with the unaided eye. In this state they fasten onto the wall of the intestine and live for years.

Symptoms.—When we consider that several hundred hookworms may live in the intestine at one time and live there for from six to ten years, sapping the blood, wounding the intestine and poisoning the body, it is not strange that the body becomes diseased, for in this disease much blood is lost. In the severe cases of the disease, the blood is reduced to one-fourth to one-sixth of what it should be. Perhaps the best concise description of the symptomatology of this condition is expressed by Farrell.[12] In such cases we find the victim's normal color replaced by a pale, sallow complexion; the lips are pale, the mucous surfaces generally are pale, and the skin is of a pale, yellowish hue. The eyes are listless, the pupils dilated and not very responsive to light; often they present a blank stare, fish-like in character. The hair is dry and scant, especially in the armpits. The face and the ankles are often swollen, anemic ulcers frequently appear on the legs, and the abdomen is prominent, giving rise to the term "pot-belly." The chest is flat, and the shoulder blades stand out prominently, suggesting "angel wings." When the disease occurs during the growing period there is a marked retardation in development; that is to say, a boy or girl may not be developed at 18 beyond what would be expected of one at 13 or 14 years of age. The appetite is often perverted, so that the sufferer has a craving for a particular kind of food, and often for certain substances not foods. For example, victims of the disease frequently crave clay, and for this reason are termed "dirt-eaters." Again, coffee-grounds or salt may be the substance desired.

The intestinal wall is considerably damaged by the worms, and becomes tender to pressure especially over the pit of the stomach. Where the worms bite, raw surfaces are left, so that it is easy for any germs, such as may cause typhoid fever or tuberculosis, to get into

the body and set up a disease more violent in character, and more frequently fatal, than hookworm disease. There may be severe headaches, lassitude, dizziness and inability to sleep. The heart is poorly nourished by the impoverished blood, yet it is called on to do the work necessary to keep the body supplied with oxygen from the lungs and food from the digestive tract. As a result the heart's action becomes labored, so that hookworm disease is frequently mistaken for heart-disease or Bright's disease. Most of such cases can easily be cured by getting rid of hookworms.

It must not be inferred that every person who is infected with hookworms suffers with all the symptoms mentioned. Much of the infection is so mild that the presence of the disease might not be suspected. In cases of medium severity, one or more of the symptoms will be present, but the existence of the disease cannot be confirmed until the eggs of the worms are demonstrated in the excreta by microscopic examination.

The disease, however, even in very mild cases, is a menace for two reasons: First, any infection exerts a handicapping influence on the victim. This has been shown among students who, though mildly infected, were underdeveloped in size and were backward in their studies; and, being below the standard, they were more subject to other diseases. Second, the persons mildly infected are carriers and distributors of the hookworm eggs, and may become responsible for the disease in a severer form in themselves and in other persons.

Treatment.—Treatment of hookworm disease consists, primarily, of the administration of a drug which will kill and expel the worms from the intestine. In severe cases this is followed with a tonic to bring back some of the lost health and vitality.

Various drugs have been used to destroy hookworm. The three that stand out as most prominent are thymol, the oil of chenopodium and chloroform. The treatment as advanced by Farrell in 1914 is as follows: "Hookworm disease is usually treated with Epsom salts, and with powdered thymol given in capsules. The object of the Epsom salts is to free the intestine from mucus or other substances surrounding the hookworms and protecting them from the action of the thymol. The patient should take little or no supper on the evening before the thymol is to be administered. As early at night as is convenient he should take a dose of Epsom salt. The next morning as early as the salt has acted, half the number of capsules of thymol prescribed for the whole treatment should be taken. Two hours later the remaining capsules should be taken. Two hours after the second dose of thymol, another dose of Epsom salt should be taken, which will expel the

hookworms that have been forced to loosen their hold on the intestinal wall by the action of the thymol, and will also get rid of the excess of thymol before it has had time to produce any harmful effects on the patient. Nothing should be eaten on the day the capsules are taken until the final dose of Epsom salt has acted well. A little water or strong coffee, without milk, should alone be allowed.

As alcohol and oils dissolve thymol, making it actively poisonous to the patient, the use of them in any form would be exceedingly dangerous. Gravy, butter, milk, all alcoholic drinks and patent medicines, which generally contain alcohol, should be forbidden on the evening before and on the day of the treatment. Moreover, as many hookworm patients have dilated stomachs which do no readily empty themselves and it is important that the thymol reach the small intestine at once, the patient should lie on the right side for at least half an hour after taking each dose of thymol.

Dose of Thymol.

Age, Years	*Grains*	*Grams*	6 A.M.	8 A.M.
1 to 5	7.5	0.15	½ dose	½ dose
5 to 10	15.	1.	½ dose	½ dose
10 to 15	30.	2.	½ dose	½ dose
15 to 20	45.	3.	½ dose	½ dose
20 to 60	60.	4.	½ dose	½ dose
60 and upward	45.	3.	½ dose	½ dose

The dose of thymol varies with the age of the patient. As the disease retards development and persons 18 years old often have only the normal growth of 13, apparent and not actual age determines the dose. A competent physician, of course, should supervise the treatment. The accepted scale of doses is shown by the accompanying table.

The thymol is powdered and given in capsules. If sugar of milk is added grain for grain, the thymol operates better.

In a majority of cases two treatments like the one just described will expel all the worms. In 1518, out of 3630 patients treated in Porto Rico, a single treatment effected a cure; a second treatment was sufficient in 1166 cases; 518 required a third; 247 a fourth, 104 a fifth; 47 a sixth, and so on until the last case was freed from hookworms by the eleventh treatment. Frequently the worms not killed by the thymol are sickened to a degree that they do not deposit any eggs for approximately two weeks. By a microscopic examination, made two weeks or longer after the last treatment, it is possible to know

when all of the worms are destroyed, and the treatment completed. When a microscopic examination is not possible, the feces expelled by each treatment can be examined for hookworms in the manner already described. When no more worms are seen, one extra treatment for good measure should be given."

A few years ago oil of chenopodium came into favor in some parts of the United States as a remedy for hookworm, and then rapidly supplanted all other remedies. It is made from a common weed, usually called Jerusalem oak, or goose-foot, and is therefore very cheap and the supply inexhaustible. It is more effective than thymol and is if anything, less dangerous to the patient. To correct the constipating effect of the oil of chenopodium, Hall and Foster strongly advise giving castor oil with the chenopodium, and also afterward. This gives a maximum of both efficacy and safety. The usual method of giving oil of chenodium is 5 to 15 drops at two hour intervals, each dose being accompanied by castor oil.

Of late chloroform has been given with chenopodium in which it dissolves. Hall and Foster have claimed, by means of extensive experiments on dogs, that chloroform itself is more efficient against hookworms than any other drugs with which they have experimented. but carefully checked up experiences show that chloroform itself clears away but about 30 per cent. of the worms, while chenopodium is curative in at least 99 per cent. of the cases. Darling, Barber and Hacker[13] administered the oil of chenopodium, chloroform and castor oil, the dosage of oil of chenopodium being 30 millimeters in one series and 40 millimeters in another. They found that the addition of chloroform with the oil of chenopodium, particularly when in considerable amounts, enhanced the danger of vomiting, and in that way rendered the combination less efficient. They have concluded that the maximum dose 0.5 cubic centimeters three times, or 1.5 cubic centimeters is the treatment for recommendation as a routine vermicide. This did not have toxic effects as in the full dose and the treatment gave very satisfactory results in removing 90 per cent. of all worms present. If chloroform alone is used, it should not be repeated in less than three weeks, since it does some temporary damage to the liver which may not be completely repaired in less than that time.

Prevention.—Methods of prevention of hookworm disease are now being carried out by the people in the southern climates by wearing shoes, in that way preventing the entrance of the hookworm larvæ through their feet. Kneeling bare-kneed or resting the bare hands on the moist ground beside a stream or pool to drink, drinking water which is directly or indirectly polluted, dirt eating, eating with soiled

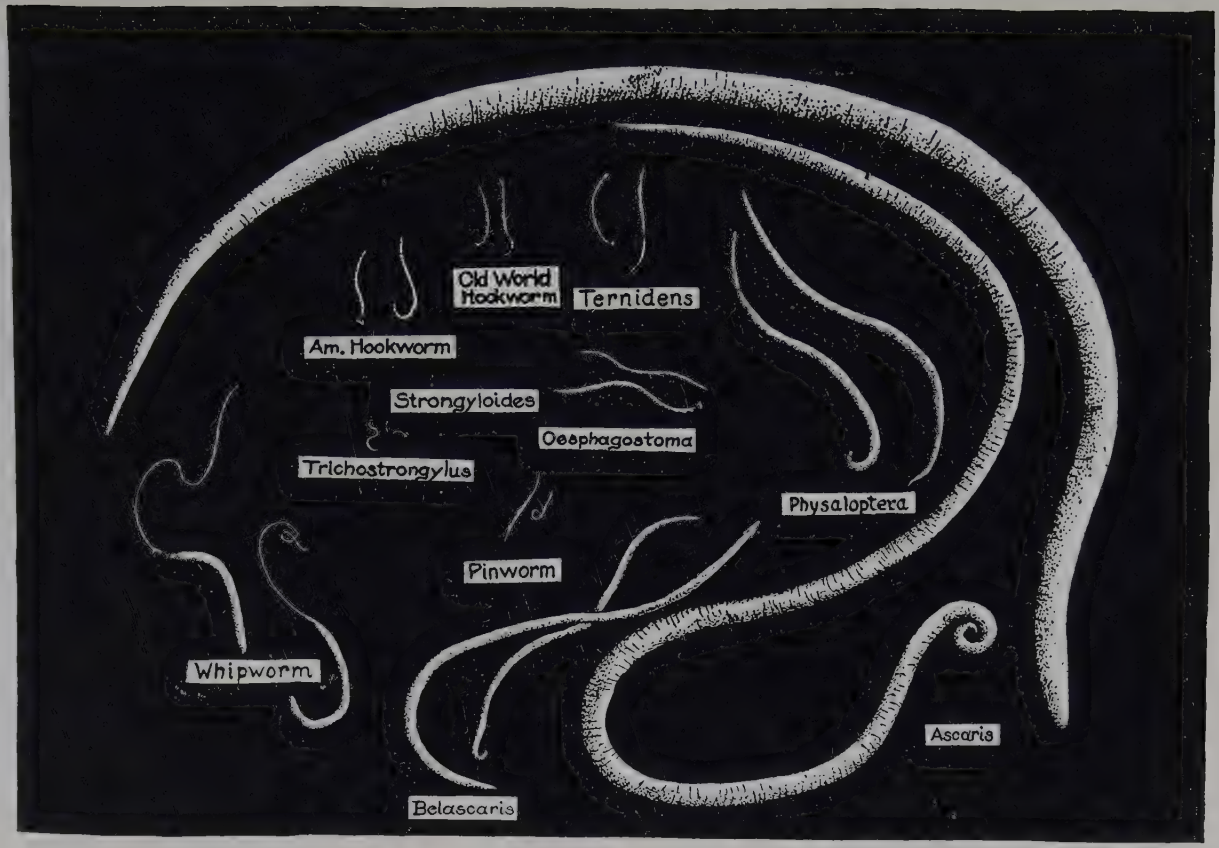
hands, the chewing of dirty finger-nails are advised against. By these and a hundred other ways the laborer who had become infected is now protected. If it were not for the pitiful ignorance and stupidity of the people to be dealt with, the prevention of this disease would be easy, but the hookworm has a valiant ally in the stunted brain and will of its victims and in the unsanitary habits which characterize almost every hookworm infested country. Thus the prevention and eradication of hookworm disease is the prevention of pollution of the soil—in other words, proper sanitation. Not only the hookworm, but almost all of the true nematode parasites of the human intestine are the direct outcome of unsanitary conditions. Rural communities require privies in connection with each home. Their use should be restricted to the women and the children or to the family of the manager. When more privies or latrines of some sort, even if it be only a ditch, are used by the rural population, hookworm infection will soon pass away. As was stated before, the worm lives but about a year, and thus this point of sanitation is important.

The isolation and treatment of infected persons is to be highly recommended, especially in cases of immigrants or new arrivals from infected regions. It is to the great credit of this country that so much work has been done in this infection, and the work done by the American Hookworm Commission, financed by a gift of a million dollars from John D. Rockefeller, which is now extended throughout the West Indian Islands, Central America and Egypt, requires the greatest commendation and thanks.

INTESTINAL ROUNDWORMS.

The so-called intestinal nematodes are frequently met with, especially the Ascaris lumbricoides and the Strongyloides. Such infections are met with in people of the North and city dwellers, as well as in those of the South and tropical countries in whom they are more numerous. For a long time it was believed that these infections were not capable of producing symptoms, or that such that were, were very slight, and therefore their significance was almost negligible. However, severe symptoms may occur in connection with them and they should always be looked upon in a pathogenic sense.

The presence of intestinal worms of practically all species can be determined by the finding of eggs in the feces. For this purpose, a small portion of feces about the size of a walnut, is mixed with about 50 cubic centimeters of distilled water, drained through several thicknesses of wide mesh (surgical gauze), and centrifuged at high speed



Intestinal nematodes of man, natural size. Male and female of each species shown, except Strongyloides, in which only the female is known. The female of Esophagostoma is immature, the mature form being unknown. (*Chandler.*)

for about ten minutes. The upper liquid is then poured off, more water is added, thorough shaking is accomplished and centrifuging again. The material shown down at the bottom of the tube contains the eggs and can be readily found under a microscope. The eggs are colored yellow or brown from bile in the feces. The eggs from hookworms, Strongyloides and a few others are clear and colorless. In the instance of pinworms, the adult female containing the eggs usually passes out entire, whereas in Strongyloides, the eggs hatch before leaving the host.

Treatment.—Since the treatment of most of the nematodes in general is similar, it may be dispatched here. It is important that certain general principles be carried out, because almost all anthelmintics are poison and the host is simply protected because of their comparative insolubility or their rapid elimination. For worms situated in the upper portion of the digestive tract, drugs such as chloroform which is rapidly absorbed and eliminated can be used, whereas for worms situated in the lower portion of the digestive tract, insoluble drugs would in general be better. Certain drugs have more or less specific action against certain species of worms, as evidenced by the use of oil of chenopodium against Ascaris, and chloroform against hookworm. This apparently specific action may be due to a mode of life of the worm affected which makes it particularly easily reached by the drug. In the case of hookworm, for instance, Hall and Foster believe that their specificity may be due to the fact that hookworms are blood-suckers, and that the chloroform rapidly absorbed by the blood is ingested by the hookworm in amounts sufficient to cause stupefaction or death.

Santonin has been the classical drug for expelling Ascaris, but oil of chenopodium has recently been demonstrated to be considerably more effective. The oil when properly administered is almost 100 per cent. effective for Ascaris, and is more dependable than any other drug commonly used for worms. The oil of chenopodium, administered as for hookworm, is probably the most effective remedy with the whipworm, which is usually quite resisting to treatment. In the instance of pinworms, on account of their situation in the lower part of the intestine, drugs which are not rapidly absorbed from the intestine and relatively insoluble are the best. Of these thymol, male-fern, and best of all, oil of chenopodium, are effective remedies. The treatment for the Strongyloides is the same as that for hookworm, although they are quite difficult to expel. Preventive measures against all the true nematode parasites of the intestines consists mainly of proper sanitation.

ASCARIS LUMBRICOIDES OR EELWORM.

This is one of the largest nematode parasites known, the female averaging about 10 inches in length, and occasionally measuring a

Fig. 136.—Ascaris—dissected to show anatomy—female above, male below. (*Chandler.*)

foot and a half, while in diameter the body may be as large as an ordinary leadpencil. The males are usually several inches shorter. The infection with these worms is very common (occurring in all parts

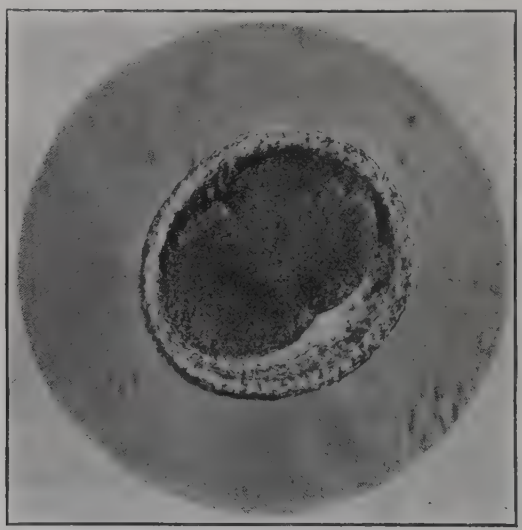


Fig. 137.—Eggs of Ascaris lumbricoides ×500.

of the world, and especially in children) and is said to have been found as far north as Greenland and Finland. In the tropics they are abundant and almost universally present in children, each individual harboring anywhere from two or three to several hundred worms. The

Ascaris can be immediately recognized by its large size and robust form, and by it being more slender at the head than at the tail. The sexual organs occupy the greater part of the body. The human species is usually looked upon as a variety which occurs in hogs.

The life history of Ascaris is usually thought to be very simple. The eggs, of which thousands are deposited by a single female, develop within the egg-shell outside of the human body, in water, soil or manure piles. These eggs are about 0.06 millimeter long by 0.04 millimeter wide, elliptical in form, with a thick transparent, shell, usually bile-stained, covered outside by irregular albuminous coats which give them a rough warty appearance. In about a month or six weeks after being passed, and under favorable conditions in soil or water, the embryo will develop, which then can be seen rolled up within the shell. The egg may remain for months or years in this condition. The use of common feces as a fertilizer undoubtedly results in a wholesale contamination of vegetables and other garden products.

When swallowed by a suitable host, the hard shell of the egg is dissolved off and the parasite is liberated in the small intestine. After about five or six weeks, maturity is reached and the production of eggs begins again. The rat is very susceptible to this worm and probably is a marked factor in the dissemination of this infection.

Symptoms.—The symptoms produced by Ascaris infection vary greatly with different individuals. In some cases practically no ill effects are noted, in others there may be peculiar mental and constitutional ailments occur, such as feverishness, anemia, restlessness, epilepsy, insomnia and delirium. Stomachache, loss of appetite, nausea and nervous phenomena are commonly seen. The nervous and constitutional effects are probably due to toxic bodies produced by the worm. These worms occasionally creep forward into the mouth or nose, sometimes creep out of the intestine and body cavities, going through the intestinal wall, and may give rise to the formation of abscess.

WHIPWORM.

With the exception of hookworm, the whipworm Trichuris trichiura, is the most common intestinal worm parasitic in man. It is a nematode related to the trichina worm in which the anterior end of the body is drawn out into a long filament like the lash of a whip. The female whipworms, which are always the more numerous, are about two inches long, while the males are a little smaller.

The human whipworm is found in almost every part of the world, but is specially prevalent in warm countries, where it parasitizes both

man and monkeys. It makes its home in the cecum and may be found in the appendix or large intestine as well. When in the intestine it is said to transfix the wall with its thread-like anterior portion, but there is some evidence to show that it merely buries its long head and "neck" between the folds of the intestinal wall.

Usually the only evidence of the presence of whipworms is the appearance of the characteristic dark-colored, barrel-shaped eggs in the feces. These eggs develop in water or moist soil, in which they may live for years without losing their vitality. Infection, as far as known, occurs as in the case of Ascaris, the worms attaining a maturity and producing eggs in less than a month after the eggs have been swal-

Fig. 138.—Human whipworm, Trichuris trichura. (Trichocephalus dispar.) ×15.

lowed. Although the whipworm feeds on blood, it undoubtedly produces toxins, as evidenced by the increase in eosinophiles in the blood which nearly always occurs in case of whipworm infection and by the occasional mental disturbances and other nervous symptoms.

PINWORMS.

One of the most frequent and widely distributed intestinal parasites of man is the pinworm, Oxyuris vermicularis. It is seen almost universally in children at one time or another in temperate as well as in tropical countries. It inhabits the lower part of the small intestine and the cecum.

The adult females are whitish worms, about two-fifths of an inch in length, and have the diameter of about an ordinary pin. The males are only about half as large and have the posterior end of the body

rolled ventrally. The adult females filled with eggs leave the small intestine and cecum and wander back to the rectum whence they are passed out with the feces or creep out of the anus, especially in the evening or at night, causing intense itching. These egg-filled females, or free eggs which already contain coiled embryos, live in the moist groove between the buttocks, in girls sometimes creeping forward to the vagina. From the scratching and rubbing which results from the itching in the vicinity of the anus, the fingers and fingernails become infected with the eggs. In that way they may be carried directly or indirectly into the mouth, thus causing reinfection, or they may be transmitted from person to person.

Fig. 139.—Early development of pinworms, Oxyuris vermicularis. *A*, Newly laid egg containing tadpole-like larva. *B*, Egg 12 hours later with nematode-like larva. *C*, Egg with fully developed embryo. *D*, Newly hatched embryo. ×500. (*A* and *B* after *Braun; C* and *D* after *Leuckart.*)

After infection, which probably nearly always occurs by way of the mouth, about two or three weeks elapse before sexual maturity is again attained and the eggs and females reappear in the feces.

As a rule no inconvenience is felt from the presence of even large numbers of pinworms, yet they sometimes produce reflex nervous symptoms, probably by secretion of toxins, and they may interfere with the normal action of the bowels. It is believed that they are also capable of producing lesions in the appendix which may culminate in appendicitis. The intense itching which they produce by creeping in the vicinity of the anus is usually the most disagreeable effect of their presence. They are most successfully removed by enemata methods of treatment.

STRONGYLOIDES.

Another parasite of the intestine which is of wide distribution and locally very common is Strongyloides stercoralis, a very small worm about one-tenth of an inch in length which bores deep into the mucous membrane of the intestine. The female Strongyloid, which is the only sex known, can be recognized by its small size, and microscopically by the chain of six or eight eggs, lying near the middle of

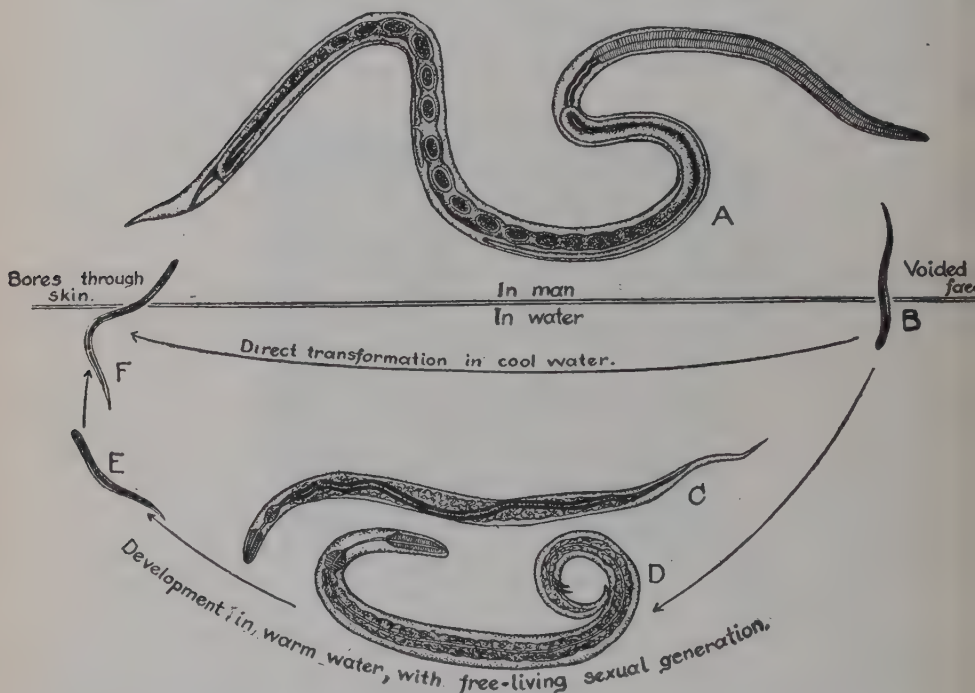


Fig. 140.—Life history of Strongyloides stercoralis. *A*, Adult female in intestine (note long pharynx, egg-containing uterus and vaginal opening on posterior third of body. *B*, Newly born embryo as passed with feces. *C* and *D*, Adult female and male, respectively, of free-living generation. *E*, "Rhabditiform" larva, from female of free-living generation. *F*, Filariform larva, resembling grandparent, and formed by metamorphosis of *E*, ready to infect by boring through skin. ×75. (Partly after *Looss.*)

the body, visible through the delicate body wall. The eggs, which are deposited deep in the intestinal coat, normally hatch before leaving the digestive tract of the host and grow considerably, so that when the feces of an infected person are examined microscopically the active writhing larvæ 250 in length, can be seen darting about in snakelike fashion. Further development of the larvæ takes place in water of

fairly high temperature, under which conditions it attains a sexually mature form, male and female. They now copulate, and the females lay 30 or 40 eggs, all within a couple of days. This second generation of eggs hatch into tiny free-living larvæ, resembling the parents, but after their first moult they lose the parental characteristics and become like their grandparents, after which they soon die unless they gain entrance into the digestive tract of the human being.

The method of infection is similar to that of the hookworm. While the larvæ may occasionally gain entrance to their host with polluted water or food, they are able to bore through the skin as do the hookworm larvæ.

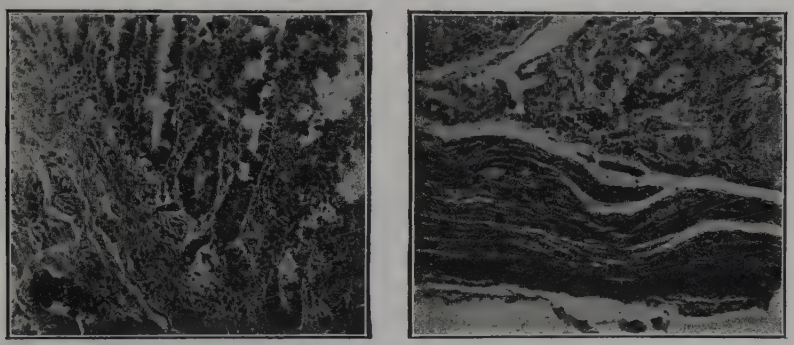


Fig. 141.—*A*, Larvæ breaking through into the muscularis mucosa. *B*, Just inside outer longitudinal muscular coat of the duodenum. (*Gage.*)

The most characteristic factor of this nematode is its ability of boring its way through the various tissues of the body. Gage has reported a case of an emaciated individual with a persistent cough who was supposed to have tuberculosis, and on the examination of his sputum, the larvæ were noted. At *post mortem* the mucus from the duodenum contained large numbers of adult worms in all stages of development, these being also found in the lungs.

In the majority of cases, however, the Strongyloides do not cause very serious ill effects from their pursuit of life and happiness in the intestine. Nearly all cases of diarrhea and dysentery in which the Strongyloides were formerly supposed to be the chief agent, can now be ascribed to some other cause. Barlow, however, reports five uncomplicated cases of intermittent diarrhea without blood or mucus in the stool, in which there were colic and certain nervous symptoms, and I have seen such cases.

OTHER SPECIES.

There are numerous other species of nematodes which occasionally make their home in the human digestive tract. Some are locally common, others merely sporadic in their occurrence. Stephens lists 59 species of nematodes as having been observed in man. None of these are of great importance since they seldom cause serious ailments and most of them are rare. The following is a list of the names of some of these:

Belascaris cati
Toxascaris limbata
Physaloptera mordens
Trichostrongylus (several species))
T. instabilis (subtilis))
T. orientalis
Ternidens (or Triodontophorus) deminutus
Esophagostomum apiostomum (brumpti)
O. stephanostomum var. thomasi
Gigantorhynchus hirudinaceus (gigas)
Echinorhynchus hominis
Hormorhynchus (or Echinorhynchus) moniliformis
H. clarki

HILL DIARRHEA.

This condition has been described by Crombie who had abundant opportunities for studying in Simla, and the more chronic cases in patients returning to Calcutta with the disease from various hill stations.

Symptoms.—The most essential feature of this form of diarrhea is that it is limited to the early hour of the day, beginning about 3 to 5 A.M. and rarely continuing after 11 A.M., but recurring at the same time each day. From one to six motions may be passed daily, but usually from two to four, while a noteworthy feature is that not only are they unattended by pain or depression of the pulse, but they actually afford relief to the abdominal discomfort and distended feeling which precede the evacuations. The stools are liquid, frothy, and of a light-grey color, resembling whitewash, while their odor is particularly feculent, but not offensive. During the remainder of the day the patients go about their occupations without any discomfort or anxiety.

For some time the general health is but little affected. Associated with the condition, however, is usually a flatulent dypepsia, affecting both the stomach and intestinal canal which may commence immediately on the patient arriving in a hill section and therefore must rather be due to physiological processes than to any infective agency. The almost entire absence of bile from the motions point to deficient action of the liver.

Diarrhea may continue in this way for many months, and in some cases recur during each season in the hills for a number of years

without the general health being materially affected, but more commonly some degree of emaciation results. Usually when the patient returns to the lower levels, the symptoms disappear, a re-establishment of normal function takes place and improvement sets in both locally in the abdomen, and generally in the body. Generally it is met with in the height of the rainy season, declining again at the end of the rain. This distribution is of importance in connection with the possibility of the disease being a water-borne infection, for it is most prevalent just when the water-supply is purest after the hills have become thoroughly washed free of any accumulation of dust and dirt. However it has been observed that those who drank boiled water did not escape the disease. Children under twelve years of age are rarely attacked. It might be due, some believe, to individuals living on the hot plains for a while, then coming up into the hills in a debilitated condition due to long exposure to heat, when they are suddenly exposed to a great deal of atmospheric pressure which could produce sickness and other physiological perturbations.

The prognosis is good as long as emaciation has not advanced, and removal from the hills is possible after treatment has failed to control the disease. It is only neglected cases running on into sprue that become dangerous to the health.

If seen early, the treatment is very effective. Grant suggests a dose of blue pill and Dover's powder at bed-time, and in the morning an ounce of castor oil with a little laudanum. Farinaceous diet and confinement to the house for a few days then suffices to stop the trouble. For recurrence the treatment is repeated. Warm clothing is ordered and a careful diet observed. Opium should only be given with ipecacuanha, as it otherwise checks the liver secretion. The giving of fairly large sized doses of pepsin two hours after meals has been beneficial in cases. Severe cases will require peptonized milk, eggs, boiled fish and then fowl may be added as improvement sets in. Protection of the bowels from chills by means of a flannel binder round the abdomen, especially at night, is an important safeguard against attacks and recurrences of the disease. The success of these simple measures and the pepsin treatment support the physiological view of the causation of the disease.

References.

[1] Smithies, Frank: "Protozoic Enterocolitis."
[2] Sistrunk, W. E.: Jour. Am. Med. Assn., Nov. 4, 1911, vol. lvii, No. 19.
[3] Lyon, M. W., Jr.: Jour. Am. Med. Assn., Feb. 1, 1919, vol. lxxii, No. 5.

[4] Orche, Lieutenant W.: Lancet, 1917, cxcii, 297.

[5] Phillips, Llewellyn Powell: "Amebiasis and the Dysenteries."

[6] Rogers, Leonard: "Dysenteries, Their Differentiation and Treatment."

[7] Chase, Arthur F.; Tasker, Arthur N.: Jour. Amer. Med. Assn., May 26, 1917, vol. lxviii, No. 21.

[8] Chandler, Asa C.: "Intestinal Flagellates and Ciliates," p. 125.

[9] Fowler: Brit. Med. Jour., April 14, 1906.

[10] Stiles, C. W.: New York Med. Jour., 1903, vol. lxxviii, p. 877.

[11] Mertens: Berl. Klin. Wchnschr., 1892, No. 44, 1099.

[12] Farrell, John A.: Jour. Amer. Med. Assn., June 20, 1914, vol. lxii, No. 25.

[13] Darling, Samuel T., Barber, M. A., Hacker, H. P.: Jour. Amer. Med. Assn., February 23, 1918, vol. lxx, No. 8.

CHAPTER XIX.

Rectum and Anus.

PROCTITIS.

(Chronic Proctocolitis, Coloproctitis.)

Chronic inflammation of the rectum in the majority of instances involves the upper portion of the large bowel as well. It is rare that chronic inflammation is confined to the mucosa of the rectum, because in most instances it descends from the descending colon, involving the mucosa of the rectum by extension. It is met with under two forms, the atrophic and hypertrophic. Not infrequently both types of inflammation are present at the same time in different areas of the mucosa.

The atrophic form, usually chronic in nature, is due to extension from the descending colon beyond the pelvic colon into the rectum. It is more frequently seen in men and can be noted by the mucous membrane having a mild degree of congestion, somewhat dusky red in color, dry and glazed in appearance, often with adherent particles of dried feces, the rectum being very much dilated, the anal tissues ofttimes being relaxed and giving one the impression of a partition thinner than normal between the external and a largely dilated rectum beyond, occasionally with a contracted and irritable sphincter, and a patient in whom the history of constipation is distinct.

The hypertrophic form, which is supposed to be secondary to an acute variety, in my opinion is an entity beginning as a chronic process. It often involves the entire colon, is frequently met with in women, the mucous membrane being pale and swollen; there is present tenacious mucus, which mucus secretion is marked, the whole rectum giving the appearance of being smaller in caliber, due to extension of the process into the muscular and perineal tissues, and a person in whom the element of constipation may or may not be present.

Etiology and Pathology.—The atrophic form is supposed to result from chronic constipation and the habitual use of purgative medicines. Other causes mentioned are the repeated use of bulky enemata, over-indulgence in highly seasoned foods, alcoholic beverages, and so on.

In my opinion, the condition is practically always secondary to a status of chronic excessive intestinal toxemia in which the mucous membrane takes on an atony of the superficial cellular elements due to the effect of bacterial toxins upon it. Therefore it is oftenest met with in instances of the indolic or mixed forms of chronic excessive intestinal putrefaction, and this in my opinion represents an etiology far more important than the first mentioned.

The etiology of the hypertrophic form is less obscure and difficult to trace. It undoubtedly is due to the saccharo-butyric type of chronic excessive intestinal toxemia and therefore is usually made worse by the taking of large quantities of starchy foods, as bread, oatmeal, rice and potatoes.

The pathology of the two forms of chronic proctitis is well indicated by their names. In the atrophic form the mucosa is thin, inelastic, the crypts of Lieberkühn atrophied, and the substance between them greatly decreased. The solitary follicles appear large and prominent, due to the atrophied condition of the surrounding mucosa. Minute ulcerations may be present, particularly under particles of adherent feces. The pathology of the hypertrophic forms is exactly opposite. Here the mucosa will be found thickened, the follicles of Lieberkühn hypertrophied, and the interglandular spaces increased, both in depth and width.

Symptoms.—The symptoms of the two forms of chronic proctitis are for the most part identical, with the exception that while in the atrophic form constipation is a feature, in the hypertrophic form it may not be. Usually the onset is imperceptible to the patients, and in my opinion such symptoms as are present, such as impaired appetite, pale coated tongue, loss of weight, dull headache, sallow complexion, etc., are due to the intestinal toxemia and not to the proctocolitis. However, pruritus may be a feature of both varieties. In the atrophic the perianal tissues are comparatively tender and easily cracked or fissured by handling, whereas in the hypertrophic variety the discharge from above causes more or less moisture of the parts with a thickening of the anal folds which may be distinctly hypertrophied and even edematous.

The diagnosis presents no difficulties since it is made in connection with the diagnosis of chronic excessive intestinal toxemia, the examination of stool and urine specimens, plus the use of the proctoscope.

Treatment.—The treatment of these conditions is entirely dependent upon the type of intestinal toxemia present, plus some local measures. As a rule local measures are not necessary if the intestinal

toxemia is well handled. Subject matter in connection with the dietetic and bacterial treatment of chronic excessive intestinal toxemia will be found in the chapter under that heading.

Where distinct local symptoms are present the use of castor oil, once every four to seven days, or perhaps the use of magnesium sulphate in the morning, answers a good purpose in bettering the local condition of the gut. What cannot be accomplished with these can never be accomplished by the use of enemata or irrigations, however administered and whatever the solution used.

Occasionally direct application through the proctoscope may be called for. Hot irrigations of boric acid, or weak (1:500) carbolic acid solution, are of some benefit in relieving congestion, and promoting the absorption of inflammatory products as well as preparing the part for further medication. Where ulceration is distinct the coating of the interior of the rectum with calomel or equal parts of calomel and bismuth subnitrate is an excellent means of local treatment. A solution containing non-alcoholic fluid-extract of hydrastis and iodoform has been recommended. When stimulation is required, the silver salts are the best agents. The nitrate is the most generally efficacious of these; from 1 to 5 per cent. solutions topically applied often work most beneficially.

HEMORRHOIDS.
(Piles.)

Etiology and Classification.—This condition has been described since Moses' day and is found mentioned in the fifth book of the Old Testament of the Bible, and since the days of Hippocrates has occupied a definite place in nosology.

Hemorrhoids are enlarged blood tumors caused by a pathologic condition of the hemorrhoidal blood-vessels. The movable part of the rectum is supplied by the superior hemorrhoidal artery, the terminal branch of the inferior mesenteric, while the lower part of the rectum about the anus is supplied by the middle and inferior hemorrhoidals, branches direct and secondary respectively of the internal iliacs.

Among the predisposing causes of this malady (which is peculiar to the human family) is mentioned the upright carriage of man, which means that during two-thirds of the twenty-four hours superimposed upon the middle and inferior hemorrhoidal vessels are columns of blood which causes more or less distention, this being a factor because of the looseness of the mucous membrane and the abundance of the cellular tissue to which the blood-vessels of this region

ramify. Anything which prevents the portal vein from emptying itself freely into the liver must result in damming back the column of blood into each of its several radicles, thus producing engorgement and congestion. During congestion, the portal vein and its tributaries are distended with blood and it is reasonable that if there is a pathological condition of the liver, spleen or heart, it might result in local congestion of the part and thus be considered a cause of hemorrhoids.

Hemorrhoids is a wide-spread condition affecting a considerable proportion of adults. Heredity does not seem to play any part, nor do matters of race. Excesses of eating and sedentary habits are undoubtedly factors. Pregnancy and parturition are factors in the female, as is probably excessive indulgence in stimulants, such as coffee, alcohol, etc.

Exciting Causes.—A number of factors are mentioned as exciting causes among which are, constipation, and the act of defecation in which excessive straining is engaged in. Thrombotic external hemorrhoids are often a direct result of the passage of costive stools, the straining causing a rupture of a small vessel at the anal rim with a tumor formed by the escape of blood. The habitual use of purgatives and medicine is one of the most noteworthy of the exciting causes, this bringing about a congestion and local irritation, and a violent peristaltic straining excited. As is well known, colocynth, jalap and aloes have a local congestive effect upon the lower rectum. Diseases of organs adjacent to the rectum in which there is a damming back of the circulation, and in liver and valvular heart conditions chronic congestion of the inferior hemorrhoidal veins may take place and cause the development of hemorrhoids. Tight lacing has been mentioned as a cause of hemorrhoids, but probably is rather far-fetched.

Classification.—Hemorrhoids are divided into two classes, external and internal. External hemorrhoids are situated below or upon the distal side of the mucocutaneous line and are covered by the skin or skin and mucocutaneous tissue of the anus. They involve the vessels of the inferior venous plexus, which is connected with the general systemic circulation, and are fixed in their position outside of the external sphincter muscle.

Internal hemorrhoids are located above or upon the proximal side of the mucocutaneous line, invested wholly by mucous membrane, implicate only the vein of the superior hemorrhoidal plexus, which is connected with the portal system, and when accidentally prolapsed are capable of being returned to their normal habitat within the rectum.

Various other classifications are engaged in such as bleeding, itching, protruding, etc. External hemorrhoids are for practical purposes, further divided into varicose external hemorrhoids, thrombotic external hemorrhoids, and skin tags or connective-tissue pile.

EXTERNAL HEMORRHOIDS.

External varicose hemorrhoids are due to engorgement of the veins surrounding the anus and generally are associated with some inflammatory trouble and relaxation of the skin. Commonly they are associated with spasmodic conditions of the sphincter and perhaps a fissure. As a rule no special local symptoms are caused by their existence, although occasionally they may rupture and bleed causing slight ulceration.

The diagnosis of this condition can be made by a visual inspection and the treatment is simple. Where a spasmodic condition of the anus exists, gradual dilatation may be necessary. Usually this can be accomplished by means of anal dilators, the best lubricant being about two drams of suprarenal extract to an ounce of vaseline. Cold applications may be used or about a 25 per cent. belladonna ointment. Hot applications are sometimes of benefit when kept up for an hour or two. The condition is simple and tends to correct itself unless spasm of the sphincter is present. It usually is found that when this spasm is present, the symptoms are due to the spasm and not to the condition of hemorrhoids.

Soft Bodied External Hemorrhoids.—These are found single or multiple, and if the blood has extravasated beneath the skin they are of a cyanotic bluish color. Sometimes they are round and other times oblong and pear-shaped. They are generally situated in the margin of the anus and due to thrombosis within the vein, or to a rupture of the vein and escape of blood into the surrounding tissues with tension.

Symptoms.—What is commonly known as a sudden attack of piles is usually as follows: The onset is abrupt and usually following the straining at stool. Generally there is a slight burning and feeling of tension for a few hours. There may be some degree of pain due to the reflex spasm of the sphincter that is set up. As a rule, however, the discomfort increases and there is a feeling of a foreign body in the grasp of the external sphincter. Examination at this time discloses a tumor which usually is exquisitely tender, and on being touched gives a peculiar burning pain. If treatment has not been resorted to, after a few days the local pain and distress disappear and one feels simply the sensation of a foreign body within the grasp of the

sphincter. This usually persists for a number of days. In from about four days to two weeks an absorption may take place, but usually relief is not afforded until the pile has ruptured, liberating its content after which the relief is sudden.

Fig. 142.—Inflamed and edematous external hemorrhoids. (*Lynch.*)

Treatment.—While the treatment for the condition is simple, there are two things to remember about it. This area of the body, particularly if the swelling has been there for some hours, is liable to become affected with a secondary edematous swelling. Sometimes it is impossible to avoid this edema, and when it is present it is quite as distressing as was the original pile which might at the time be

collapsed from incision or spontaneous rupture. It brings on a distinct spasm of the sphincter and generally takes several days to disappear. The second matter is somewhat debatable, although my opinion is rather definite.

I believe that these small tumors should be incised early, for I have found that the possibility of a secondary edema is less if they are incised within the first few hours. After a careful cleansing of the parts, from 5 to 10 minims of a 10 per cent. solution of cocaine or novocaine is injected in the most prominent part of the tumor. One of the fingers is lubricated and introduced into the rectum, passing under the base of the tumor to make it more prominent, and an incision transverse to the anal margin is made, at which usually the clot slips out, can be picked out, or pressed out by the finger within the anus. Since there is danger of the clot reforming and the oozing is often considerable, gentle packing may be done. The use of some sedative ointment and a T-bandage is all that is required because the condition heals up within a few days.

In pedunculated growths of this kind excision may be used. This is more of an operation than simple incision, and care with antisepsis is much more important. Ofttimes the wound left requires one or two sutures.

Skin Tags.—The connective-tissue external piles are common, and usually they cause no symptoms. They probably are due largely to the puckering of the skin around the anus. However there may be considerable distress due to the impossibility of keeping the parts clean, in which instance removal would be required.

Where there is any local irritation the use of hot applications or an ice-bag or gauze saturated with boroglyceride and placed over the anus, or perhaps a suprarenal extract ointment would answer all indications.

When they are well developed and a source of worry to the patient it is best to remove the tags under local anesthesia. For this purpose a few drops of a 1 per cent. solution of cocaine or novocaine is injected around the base of the tumor, these are then removed painlessly by means of knife or scissors. It is best to allow the freshly cut surface to heal by granulation.

INTERNAL HEMORRHOIDS.

Pathology.—Upon section a venous pile is found to be composed of dilated blood-vessels with a variable amount of connective tissue around and between them, the whole being contained in a covering of mucous membrane. The relative proportion of vascular and connect-

ive-tissue determines the firmness and physical characteristics of a given tumor. In all probability the tumor begins as a dilatation of the venous tufts situated beneath the mucosa at the distal extremity of the movable rectum in which the radicles of the superior hemorrhoidal vein originate. The capillaries and small veins become involved and a varicose condition with perceptible swelling ensues. Slowly a hyperplasia is established, and then with some thickening of the vessel walls and infiltration and congestion around the vessels the pile assumes its full size and form.

In the early stages internal piles remain entirely within the bowel. But as they increase in size and the constant downward effect by the act of defecation, they lengthen and push toward the anus, until ultimately they may be protruded at every stool. The number of tumors varies from one to six or seven, there being usually two or more.

Fig. 143.—Section of internal pile tumor. (*Cooke.*)

The so-called capillary hemorrhoid differs considerably from what has been described, being composed of a circumscribed mass of blood-vessels lying in and immediately beneath the mucosa. It has rather a nevus appearance, its shape usually oblong and its color bright red or purplish. In a cross section under this nevoid tumor formation the characteristic venous sinuses as seen in the pedunculated type are noted. The condition is rare, usually giving no symptoms other than bleeding, which may be quite profuse in this type of hemorrhoid.

The so-called mixed hemorrhoid is a combination of the external and internal varieties usually due to a merging of two independently formed tumors, or from an extension downward of an internal tumor.

Symptoms.—Internal hemorrhoids may exist for years without giving rise to any symptoms other than the fact that they protrude and bleed occasionally. In proctologic examinations one not uncommonly sees more or less of a hemorrhoidal condition in the lower part of the rectum, without there having been any symptoms, not even a bleeding. In the minority of cases when the growths are of some size, pain and a dull aching in the rectum takes place. This is incident upon the straining and is controlled by the reduction of the mass.

Perhaps the distress is due to strangulation which may be further complicated by an edema of the anal margin due to pressure.

The next commonest symptom is hemorrhage, which perhaps is the most conspicuous in the sense that it occurs at some time in the progress of almost every case. As a rule hemorrhage does not occur until the tumors are of some size, and then only when costive stools or scybalous masses are voided. As a rule the loss of blood is slight, consisting of a few streaks upon the stool, or a few drops following the passage. However, when the tumors reach the stage of protrusion

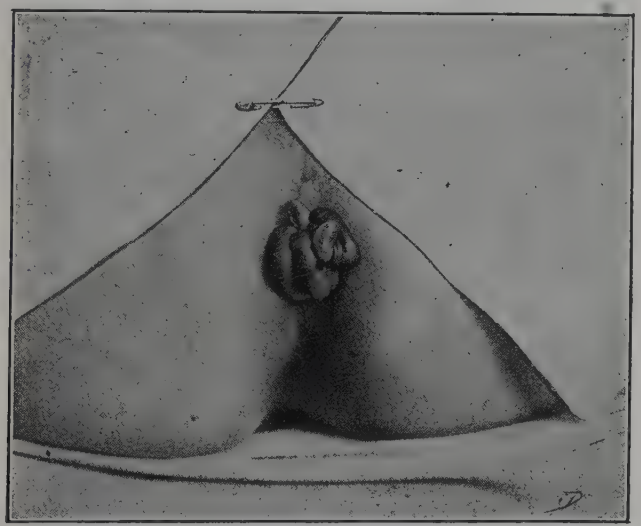


Fig. 144.—Prolapsed internal hemorrhoids, showing hypertrophied and inflamed papilla. (*Lynch.*)

and the factor of the restriction by the sphincters becomes operative, the hemorrhage may be copious. Reduction of the pile or piles within the sphincter usually controls the hemorrhage, although it is probable that more or less bleeding continues.

Protrusion is a symptom which at once absolutely establishes the diagnosis of hemorrhoids. It does not occur in all cases and not uncommonly one meets with marked types of rectal distress with bleeding and considerable tumor formation within the rectum without there having been any protrusion; seen mostly in the short stout tumors not long enough to reach through the sphincter muscles. Protrusion is best seen in the old cases in which reduction has been practised for many years. In them a relaxation of the sphincters takes place so that the tumors prolapse upon the slightest exertion. In such

cases not uncommonly walking or the erect attitude is all that is necessary to bring on a protrusion.

Mucus discharge, while not distinctly a symptom of hemorrhoids, may be present, particularly when protrusion or prolapse of the rectal mucosa exists. It is merely an expression of a chronic catarrhal inflammation of the mucosa. Commonly when mucus discharge is present, an annoying pruritus exists.

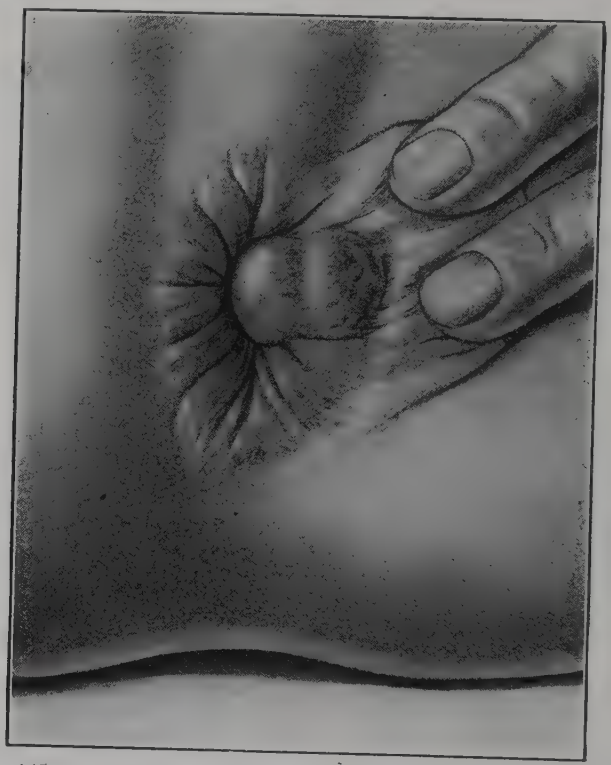


Fig. 145.—Mixed external and internal hemorrhoids. (*Cooke.*)

Symptoms which are not uncommonly found in hemorrhoids are those due to fissure of the rectum, which usually escapes diagnosis because of the bleeding or protrusion incident to the hemorrhoids. Not uncommonly one sees cases in which there is considerable distress, backache and feeling of weight in the rectum, reflex pains in the bladder, prostate, ovaries and uterus, and sometimes this pain may be referred out into the hip. I desire here to make etiological connection between some cases of persistent gastric hyperacidity and secretion and hemorrhoids. In the last ten years I have seen at least a

score of such cases in which the removal of the hemorrhoids caused a rapid and quick subsidence of a persistent gastric distress, which sometimes had been present for years.

Diagnosis.—The diagnosis of internal hemorrhoids is usually a simple matter when the tumors are prolapsed or can be made to protrude by voluntary effort of the patient. In the majority of cases

Fig. 146.—Protruded internal piles with anal edema. (*Cooke.*)

when this is not present an examination is required. As a rule one should proceed by digital examination. This is rather valueless in the early stage of internal hemorrhoids because they are small and soft, the rectum is voluminous, and the pressure of the sphincter on the finger is confusing. It is probable also that in the majority of instances the tumor mass temporarily disappears under the pressure of the examining finger. However, if the hemorrhoids have existed for a long time and thrombosis has occurred as a result of inflammation, then the diagnosis can be made by the finger alone.

As a rule direct inspection is required, which can be done by the use of a short proctoscope not much more than 10 centimeters or two to three inches in length. By this means the entire circumference of the region involved is at once exposed and the tumors can be seen protruding in the lumen of the instrument.

Treatment.—It must be remembered that in many cases of hemorrhoids some form of organic trouble exists. For this reason it is well that not only a careful inspection of the rectum be performed, but that the heart, liver and other organs be considered in connection with a hemorrhoidal condition. One must remember, too, that locally the early symptoms of carcinoma, in constipation and hemorrhage, resemble a hemorrhoidal condition, and unless an examination is made, a hemorrhoidal condition may be diagnosed and treated as such, or is assumed to be such when a well advancing carcinoma is present instead or in addition. Where the hemorrhoids are secondary to cirrhosis of the liver, well-advanced heart disease, or pelvic trouble, manifestly the treatment should be directed to these conditions as well as to such as might be necessary to relieve the patient of the local rectal symptoms. Of some importance in connection with the prophylaxis and even the palliative treatment of hemorrhoids, is that the external parts should be well cleansed after defecation, not only with toilet paper, but with cloth sufficiently dampened or wet toilet paper to keep the parts clean. Important in this connection is the matter of the correction of constipation, the cause of which must be found out in the case and proper treatment instituted. For an individual to remain constipated with constant straining at stool, the mechanical irritation of hard lumps, and the large hard masses coming through the lower rectum, is a combination for hemorrhoid formation and continuation that is ideal. Where a heart condition exists treatment should be directed to the condition of the heart muscle, rest encouraged, and treatment which belongs in the realm of cardiac therapeutics engaged in. Usually in cirrhosis of the liver when hemorrhoids exist very little worth while other than palliative treatment can be conducted. The same is true of hemorrhoids in pregnancy, but usually after parturition the hemorrhoids decrease in size and disappear, although sometimes they persist and require attention. Where there is some pelvic condition interfering with return circulation of the hemorrhoidal vein, laparotomy may be required for its elimination after which the hemorrhoids may be immensely benefited, although usually in long-standing pelvic disease where hemorrhoids have existed for years, even after the pelvic operation, the hemorrhoids persist because they have become organized.

When one considers the numerous instances of hemorrhoids that are met with in practice and the years that operative procedures have been engaged in for their cure, one is forced to conclude that the public are not willing to consider operation unless it is the only alternative. The best of proctologists teach that operation is the only positive cure and to that we must all agree, but when one considers the centuries of work in medicine for the treatment of hemorrhoids, the ever-ready wish of the average person to resort to and persist in palliative treatment or anything wherein he does not have to take a general anesthetic and resort to what he concludes in his mind is an operation, we must in virtue of the fact give way to the public to the extent of being as proficient as possible in handling these cases by other than radical operative means.

In the instance of protrusion complicated by strangulation, which not infrequently is the condition when the case is first seen, palliative treatment is required. This consists of the reduction of the strangulated pile, the use of applications of cold water compresses, a bag of crushed ice, or perhaps a hot poultice. An ointment of much value here is one containing 2 per cent. of cocaine oleate with about 25 per cent. of suprarenal extract in 1 ounce of 10 per cent. tannic acid ointment.

Various trusses have been devised for the purpose of preventing protrusion, but in my opinion when the danger of protrusion is as great as that, the injection method or radical operation are indicated.

Hemorrhage is seldom excessive enough to place life in immediate jeopardy but usually controls itself. However, rest in the recumbent position, applications of ice, and the rendering of the stool soft to prevent straining and traumatism usually fulfill all requirements.

For the purpose of controlling the pain and inflammation and local distress, various pile ointments have been devised, and also means for their introduction. Unquestionably much may be done by the use of local remedies to allay inflammation and subdue pain, but they should always be employed with the idea of palliation and not a cure. While in many instances the ointment can be introduced with the finger, the employment of some means such as a perforated pile pipe answers to good purpose. Nowadays, most every druggist has a pile pipe fitting which can be screwed on a collapsible tube containing the ointment. This is a very handy way for introducing ointment for this purpose; particularly is this of advantage because the ordinary pile pipe is too large in circumference for easy and comfortable introduction to an irritated rectum, whereas the druggist's pipe is not much

more than a quarter of an inch in diameter at the base. Of the ointments that may be used the following are recommended:

℞ Morphine sulph.	gr. x	0.65
Hydrarg. chlor. mit.	gr. xx	1.3
Ung. belladonna,		
Glycerin	āā 3ij	8.0
Vaseline	3j	30.0
℞ Orthoform	3j	4.0
Ichthyol	3ss	2.0
Ung. stramonium	3ss	15.0
Petrolatum	q. s. ad 3j	30.0
℞ Ichthyol	gr. xxv	1.6
Anesthesine	3ss	2.0
Argyrol	3j	4.0
Vaseline	q. s. ad 3j	30.0
℞ Ung. galle and opium.		
℞ Ext. suprarenal gland,		
Ext. opium	āā 3j	4.0
Ung. simple	3j	30.0

The Injection Method of Treating Hemorrhoids.—In 1871 a young physician by the name of Mitchell in this country, devised a method of injecting hemorrhoids with a solution of phenol, but not being animated by altruistic motives, this man's name and fame went throughout the world as closely guarding the secret, and for that reason there have sprung up a number of charlatans for treating cases by the injection method. There could be but one result, and that was that the method fell into immediate and total disrepute among respectable members of the profession, in which status it remained until comparatively recently. When the results of Mitchell's work were published, out of a total of some 3300 cases there were 13 deaths, these being due to liver abscesses, stricture of the rectum, carbolic acid poisoning and so forth, and then the better men, who had spoken with contempt of this class of practitioners, felt vindicated, and were brave enough to say that this record was not worth much and that it would be best to persist in radical operation. However, the important point for the average individual with hemorrhoids, that are uncontrolled by palliative means and who refuses radical operation, is that he generally will readily submit to the injection method. The fact that this method should not be allowed to be in the hands of questionable practitioners of medicine, deserves special mention. My experience with

the injection method has been most gratifying, and when you consider that not uncommonly after radical operation hemorrhoids return, and the ease and simplicity of the injection method for the cure of the average case, a full presentation of it is required. It must be remembered that details of the technique should be thoroughly mastered to ensure a proper degree of freedom from danger in its use.

The patient, previously prepared as for any aseptic surgical operation, is placed on the left side in the Sims position. As a tight or irritable sphincter is a contra-indication to the use of this method there may have to be divulsion of the sphincter for a week before operation. This might be accomplished by means of dilators so that the sphincter may be passed readily enough for manipulation within. If there are any ulcerated surfaces existing near the site of the intended injection these should be cured before the procedure is carried out. Another important point is that acutely inflamed hemorrhoids should not be injected.

The apparatus required is a short endoscopic tube, a bivalve type of rectal speculum, or if the sphincter is sufficiently relaxed, a small Sims speculum. While an ordinary hypodermic syringe will serve the purpose, one of larger size provided with goose neck or bayonet shaped extension will be found more convenient. The rectum should be evacuated and irrigated before each treatment, and the parts scrupulously cleansed at the time of the injection. The needle is inserted well into the substance of the tumor selected for the treatment and from 5 to 20 drops of the solution selected slowly injected, the needle is kept in place for a few minutes in order that the solution may have time to act. The surrounding parts should be protected from any fluids which may chance to escape upon its withdrawal, best by coating with vaseline or some bland oil. If prolapsed, the tumors should be replaced at once. A simple dressing of sterilized petroleum or other ointment is then applied on a compress of sterile gauze and held in place by means of a tight pad and T-bandage. As a rule only one tumor at a time should be treated. When they are of small size and remain above the grasp of the sphincter, two or even three may be injected at the same sitting. The treatment usually causes little or no pain and the patient should be kept in a recumbent position for about an hour after the injection. Commonly it is possible for them then to resume their ordinary duties following the treatment. The results are better, however, when prudence is observed. As a rule a tumor rarely requires more than one treatment. After an interval of from two to five or seven days, depending upon the degree of reaction, another tumor is treated, and so on until all have received

attention. It is important that the needle be inserted to the proper depth before any part of the injection fluid is forced into the tumor. If the fluid is put too superficial, near the mucous covering, ulceration and sloughing are apt to follow. On the other hand if the needle is carried too deep it may result in acute inflammation, abscess formation and perhaps general sepsis. It is due to the latter and faulty technique rather than error of judgment in the solution employed that calamities have happened. Although even with the best of technique as it is, it is practically a radical operation. A localized septic phlebitis outside of the rectum could take place with the formation of liver abscess and general peritonitis. Sherman has made a good suggestion in that the position of the patient should be on the side opposite to the tumor, and that a speculum with a slide be used so that if any fluid comes through the needle opening it will not distribute itself excepting on the surface of that pile and then only in a very limited area, or will drop down in the cavity of the instrument on the opposite side.

Many are the types of solution employed, all containing phenol in various strengths. Sherman suggests that in large and non-sensitive growths a 50 per cent. solution of phenol with sweet almond oil is indicated, whereas if the hemorrhoid is small and sensitive an 8 per cent. solution is used with the needle being carried two-thirds of the distance from the base to the tip, and about three minims of the solution being injected. If the tumor blanches, sufficient solution has been injected. If it does not, more and waiting another minute is required. In that way, until blanching has taken place, from 12 to 15 drops may be required. Various authors have used different types of solutions, these being as follows:

℞ Ac. carbolic	℥j	30.0
Zinci chlorid.	gr. viij	0.45
Ol. olivæ	℥v	150.0

—Brinkerhoff.

℞ Ac. carbolic,
Fl. ext. ergot.,
Ol. olivæ ..... equal parts

—Overall.

℞ Ac. carbolic	℥ij	8.0
Ac. salicylic	℥ss	2.0
Sod. biborate	℥j	4.0
Glycerin	q. s. ad ℥j	30.0

—Tuttle.

℞ Ac. carbolic.,
Glycerin,
Aquæ destil. ..... equal parts
—Gant.

After the injection almost any usual ointment such as tannic acid or belladonna ointment and the like may be smeared around the parts. There are various types of suppositories containing powdered opium or morphine, cocaine or suprarenal extract which may be employed if local distress follows the injection.

Local Anesthesia Methods.—Of these there are notably the ligature operation, and ligature with excision. In cases where large masses of multiple hemorrhoids require to be removed this is best done under a general anesthetic. Where the piles are few in number removal under a local anesthesia is possible. While a 1 per cent. cocaine or novocaine solution may be employed, of late quinine and urea hydrochloride because of its lasting anesthetic effect of from three to ten days, depending upon the strength of the solution injected (which means the degree of exudate or production of fibrin), has first selection in proctologic work. The weaker the solution, the greater the length of time for complete anesthesia and the shorter the period of complete anesthesia. The 1 per cent. and the 0.5 per cent. solutions give complete and immediate anesthesia without pain during injection, and within one or two minutes after injection. With the one-half per cent. solution anesthesia may last as long as ten days, whereas the 1 per cent. solution might last two weeks. This method of anesthesia is non-toxic, its effect is prolonged, and the production of fibrin with its attendant compression about the walls of the blood-vessels is of distinct advantage. The solution should always be freshly prepared, not injected too rapidly and should not be permitted to escape from the injected tissues.

With the patient in the proper position, a half per cent. solution of quinine and urea hydrochloride is injected into the tip of the protruding hemorrhoid, local anesthesia being indicated when the tissues begin to show a pearly white or blanched appearance. The infiltrated tumor is now grasped with a clamp, drawn down so that the redundant tissue becomes taut, a nick is made at the mucocutaneous junction, in which incision a string of heavy linen thread is placed and tied tightly about the mass of infiltrated hemorrhoidal tissue so as to assist in more rapid sloughing. Usually when they are small, they need not be clipped off, as they slough off in from five to seven days. If they are large, they are clipped off, leaving a sufficiently large stump to prevent slipping of the ligature and hemorrhage. A narrow strip of

gauze is now inserted into the rectum to stimulate contractions of the sphincter, and prevent bleeding, and a sterile pad and a T-binder are tightly applied to produce pressure and the patient may be permitted to get up and walk about, although it is best for him not to attend to his business for a day. After this operation some pain due to tenesmus may require an injection of morphine or some local anodyne measure. The bowels may be moved on the following day and it usually takes a week or two for the rectal wall to heal. In this way external cutaneous or internal hemorrhoids, even though quite high up, can be removed in an ambulatory way. For the purpose of this operation a conical speculum fenestrated at the side, the Tuttle forceps for grasping the pile tumor, and a blunt pointed scissors, curved on the flat, together with the necessary suture material are required.

Operations Under General Anesthesia.—Among these may be briefly mentioned the following:

Divulsion of the sphincter may be required where there is a small and tightly contracted anus, particularly where there is hypertrophy, and often is preliminary to operation for a radical cure for internal hemorrhoids. Care must be taken not to over-stretch the sphincter and bring on a condition of paralysis, which is a most distressing condition. It must be remembered in this connection that the marked degrees of sphincter divulsion that used to be engaged in were unnecessary. Do not stretch until the tone is absolutely gone is a safe rule.

Crushing Operation.—The crushing operation as conceived by Chassaignac and developed by Pollock of London of grasping the pile tumors in the jaws of a powerful instrument and destroying them, either by the pressure of the clamp or by forming a sort of pedicle of them which is then cut off, is not necessary. With the better methods at hand today this method has gone out of favor.

Next should be mentioned the ligature method under general anesthesia resembling that already described in connection with local anesthesia.

Clamp and Cautery.—This method was suggested by Cussack in 1864, and has been popularized by surgeons generally throughout the world. The principle is based upon crushing the pile tumor, amputation of the portion above the blades of the instrument, and carbonization of the stump. The consensus of opinion at the present day seems to favor its abandonment, because as a method it is unsafe and less free from complication than the ligature. It requires special instruments, and it is regarded as unsafe because it is liable to be followed by primary hemorrhages from slipping of tissue at one or other

extremity of the clamp, or imperfect cauterization, secondary bleeding also being apt to occur because of loosening of a clot from straining, vomiting, coughing or defecation.

Removal of the Pile Bearing Area.—This was devised by Whitehead of Manchester[1] in an attempt to overcome objections to older methods in use for the cure of hemorrhoids. It is doubtful if in the range of surgery there is one operative procedure more responsible for suffering and misery which follow in its wake than this method, because in at least 50 per cent. it leaves a strictured condition of the anus. It must be remembered that in all operations about the anus, primary union is seldom achieved because of the infection of the tissues, and if infection takes place, the granulating area is filled in with scar tissue which contracts, and closes up the lower end of the rectum. As a surgical procedure it is mentioned to be condemned.

General Complications Following Operations Upon the Rectum.—These are mostly the result of infection, localized or general, reflex from disturbances of innervation or traceable to direct mechanical injury to blood-vessels, muscles or nerves.

Infection.—Fistula, fissure, chronic ulceration, phlebitis, abscess and ascending infection of the portal system are all part of an infective process. They are best guarded against by ordinary cleanliness combined with proper drainage from most wounds about the rectum.

Post operative pain arises from irritation of the peripheral nerve endings or from increased tension on the anal integument from swelling, edema, and traumatism incident to operation. These are best guarded against by operations under quinine and urea hydrochloride anesthesia, or controlled by various anodyne ointments or suppositories.

Secondary Hemorrhage.—This is rarely met with in the injection or ligature methods.

Disturbances of Bladder Function are more common after general than local anesthesia and more common in men than in women, in whom the rectum and bladder are not in immediate contact. They consist mostly of temporary retention, difficult, painful or frequent micturition occurring within the first twenty-four or forty-eight hours after operation. Retention is best handled by permitting the patient to assume the upright position.

Spasm of the Levator Ani is an extremely annoying condition, and consists of painful, spasmodic contractions, coming on more or less regularly every five or ten minutes, with an intensity which is distressing. They may be due to the ligature or they may be due to

dressing or some food substance in the rectum. This is best controlled by the use of morphine in large doses, a hot sitz bath, and perhaps a strong mixture of bromide and chloral.

Gas Pains from operations under general anesthesia due to the locking up of the bowels for several days are liable to occur and are best controlled by accomplishing a free purgation in which the instillation of oil into the rectum may be advisable, with the use of some purgative measure by mouth.

FISSURE OF THE ANUS.
(Painful Ulcer.)

If discontinuity of tissue represents an ulcer, anal fissure should be considered as such. However, as met with in the anal region it occupies a class by itself. It is an ulcerative lesion situated within the grasp of the external sphincter and is characterized by intense pain during defecation. Usually the lesion is single, situated posteriorly, or anteriorly in the region of the commissure. The affection is one of adult life although cases are occasionally seen in infants and young children. Any condition which may affect the skin of the anal region such as eczema or proctitis predisposes to anal fissure. The most frequent cause of fissure is traumatism, incident to a costive stool which is evacuated with great voluntary effort, by distending the tissues of the anal region and causing a split. In a short time infection of the surface occurs and there can then be noted an elongated ulcer running in the direction of the anal canal. It is possible that a stool containing some rough or nodular mass could lacerate the lining at the mucocutaneous junction causing a fissure. The tissue here is very thin and tender and rather easily bruised. In most instances, fissure is a very painful lesion, but in the instance of it occurring when there has long been a pruritus ani or eczema, fissure may exist without causing much, if any, local trouble. Not uncommonly fissure exists in instances of hemorrhoids. It may occur during childbirth, which probably accounts for most of the fissures in the anterior location in females.

The ulcer is superficial, the edges appearing undermined on account of the spasm of the muscle which causes inversion of the mucous edges; generally it is raw and covered with blood, or it may be quite dry and pale. Later on the edges become paler and the base more indurated, being covered with granulations, mucus and pus. A true undermining of the edges takes place with little tract bleeding from the ulcer, this being due to imperfect drainage brought about by the spasm of the muscle.

Symptoms.—Because the mucocutaneous tissues of the anus are supplied with sensory nerves to a remarkable degree and in this condition there is a contraction of the contiguous muscle, a spasmodic condition of the sphincter is established, which, after a time, brings about hypertrophy to such an extent that it might act as a formidable barrier to defecation. This hypertrophy of the external sphincter is

Fig. 147.—Typical anal fissure. (*Tuttle.*)

quite uniformly found in all long-standing fissure cases. There is always some distress after a movement of the bowels, the sensation varying from a slight burning and discomfort to a sharp or dull gnawing pain. So sensitive may these fissures become that pain is felt in the anus after urination and it may radiate into the ileosacral joint, shooting down the legs or into the back. The location of the lesion, length and depth, and the length of time it has been in existence, are the chief modifying factors with respect to the severity and other characteristics of the pain. However, so harassing can this ele-

ment of pain be that the individual may be quite unfitted for his day's work after having a movement of the bowels in the morning. One not uncommonly sees in long-standing cases a marked general hyperesthesia of the nervous system, with perhaps a considerable loss of weight and general debility. It is quite remarkable, considering such a small lesion, that so much effect upon the general nervous system can be brought about.

Reflexly there is a sphincter spasm which tends to irritate the local lesion. One should in all cases of frequent urination, or so-called inflammation of the neck of the bladder, and similar conditions, as well as prostatic troubles, examine the anal region to see if fissure exists. One occasionally finds an anal fissure to account for a so-called coccygodynia, ovarian neuralgia or vaginitis.

Diagnosis.—While fissure is a form of rectal trouble in which a correct diagnosis may be made with reasonable certainty from the symptoms alone—namely the single symptom of pain—examination of the anal region is required. The detection of the lesion is not always easy because it may be of small size and hidden. Sometimes when hidden it is at the side of a sentinel pile or in one of the folds of the anal orifice. The very best way to observe its presence is to examine the patient on the left side, the buttocks being separated, or when standing on the floor with chest resting on the seat of a chair, the patient being asked to strain, which projects the tissues of the anal region, usually bringing the fissure into view. Although much distress is brought about by digital examination, not so much by pressure upon the fissure externally as when the finger is introduced into the rectum, the latter is required to note the degree of spasm and hypertrophy of the sphincter itself. Both extreme care and gentleness are necessary to a successful examination. In some cases because of the irritability of the fissure and local distress brought about by even the stretching of a finger, digital examination may be impossible or would require an anesthetic.

Treatment.—The treatment can be divided into the treatment of the early fissures—those which have not organized or become undermined at the edges—and the so-called true ulcerative or chronic type which show little or no tendency to spontaneous healing. The rules involved in the curing of fissure are rest and drainage which are essential in the acute cases to prevent them from becoming chronic.

The early cases are best treated by the non-operative method in which the first important thing to accomplish is to do away with the trauma of stool by rendering the stool soft or mushy. For this purpose one of the most successful means is by the evening use of suffi-

cient doses of compound licorice powder to which phenolphthalein, or phenolphthalein alone may answer. If for any reason it is suspected that the stool has become hard, from 4 to 8 ounces of olive oil kept for some hours in the rectum, followed by a soapsuds enema, are required.

In many instances a local treatment to be successful is dependent upon cleanliness, to which end a mild antiseptic wash and perhaps the daily use of a dusting powder such as aristol or calomel answers. When the lesion is particularly sensitive a small quantity of cocaine or orthoform is added, or orthoform alone may be used. Sometimes it is required to stimulate the granulations or to render those that are present more healthy by the touching of the ulcer base once or twice a week with nitrate of silver. Should this be done it is wise that a pledget containing a strong solution of cocaine be applied to the ulcer before the application is made. A local application of some service is the use of balsam of Peru, about 20 per cent. in castor oil. One of the very best external applications is to paint the parts each day with tincture of benzoin. Should a sentinel pile be present no kind of local application is likely to prove effective until this is removed.

Operative treatment is required in quite a proportion of the cases. Somewhat of a controversy exists as to whether preliminary divulsion of the sphincter is sufficient or whether in all instances its division should be performed. For this purpose an anesthetic is required in all instances of divulsion although a local anesthetic may answer for division alone. The latter should always be made directly across the muscle, never at an angle, and usually performed midway between the anterior and posterior commissures. Unless one is familiar with the use of local anesthesia in this type of work, it is best to use a general anesthetic such as nitrous oxide gas even for the division. This is rather important because in most instances of long-standing fissure other pathologies in the lower rectal region are met with, these perhaps requiring surgical attention. Quite important in this connection is it that in division one is not liable to cause a paralysis of the sphincter as may take place by divulsion, although if the divulsion is not carried out as was the practice years ago—namely until the resistence of the muscle could be felt no more—the danger of paralysis is minimized.

After divulsion or division of the sphincter, the edges of the fissure are examined and carefully trimmed, and an incision made through the middle of the fissure, this carried well out into the skin, which, according to Lynch, requires special attention. He states that to be successful in the operation it is required that the edges of the

skin be trimmed to prevent early healing of this part of the wound, and that it is essential that the skin incision be kept open until the mucous membrane is healed.

The wound is now packed with iodoform or plain gauze in layers to remain for forty-eight hours, redressed at the lowest angle after the patient's bowels are moved. The wound should be packed so as to heal from the bottom, preferably with about a 10 per cent. mixture of balsam of Peru in castor oil. In about a week the wound is dressed every day, then less often until it is healed.

ANORECTAL FISTULA.

Anorectal fistula is a term used for the presence of one or more chronic sinuses about the anal region, and represents a large proportion of anal affections. They are found more often among the poor than the well-to-do, and more frequently in men than in women.

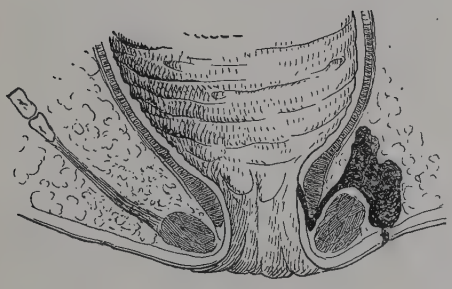
Varieties.—These are best understood by observing the illustrations, and all of them are probably secondary to an abscess leaving a chronic suppuration tract.

The blind internal fistula, while it may result from any ulceration in the anal canal, in the majority of instances is probably brought about by a cryptitis which is likely to terminate in a superficial fistula, and which if posterior in location may be deep.

External fistulæ may be superficial or deep, depending upon the location and size of the abscess cavity. In the majority of instances it is superficial or marginal. A complete fistula may result from any of the factors that have been mentioned but perhaps the most frequent cause is from an ischiorectal abscess, an abscess of the triangular space, or a deep perianal abscess.

Various other classifications of fistulæ are given, none of them serving to much practical purpose. It is important to remember that the number and location of the fistulous openings are subject to wide variation, this being particularly true of fistula which has external openings. In the horseshoe fistula the majority have their origin in a fissure of the posterior commissure which becomes infected causing an abscess in the triangular space. While it is true that the vast majority of fistulæ are not of tuberculous origin, or occurring in tuberculous people, there is no doubt that in tubercular subjects fistulæ are common. However, it must be also remembered that a fistula in a tubercular subject need not necessarily be a tubercular fistula. A tubercular fistula has rather an impenetrable wall of connective tissue, which, when examined microscopically, is undoubtedly tubercular in

Incomplete (blind external).



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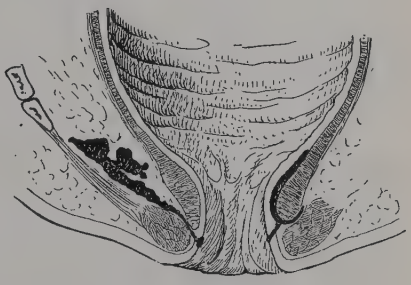


Incomplete (blind internal).

Submucous.

Fig. 148.—Types of anal fistula. (*Cooke.*)

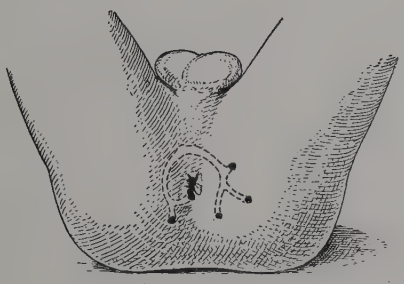
etiology. The fistula may occur between the rectum and vaginal wall, making the so-called recto-vaginal fistula, and this is generally the result of traumatism during childbirth.

Symptoms.—The symptoms of fistula proper as contrasted with those of abscess appear of minor importance. Pain is usually absent, and seldom amounts to more than discomfort in any case. There may be tenderness on sitting or on certain movements, but what usually brings the case under observation is the presence of a chronic discharge which is thin and purulent in character, offensive in its odor, and therefore distressing. This course is not true with the blind or incomplete internal fistula wherein there is more or less constant accumulation of infected material from the rectum which brings about a fistula, irritability of the sphincter, and pruritus. In the recto-vaginal fistula there is more or less of a fetid discharge from the vagina. This, or the odor attending coitus, usually brings the case under observation.

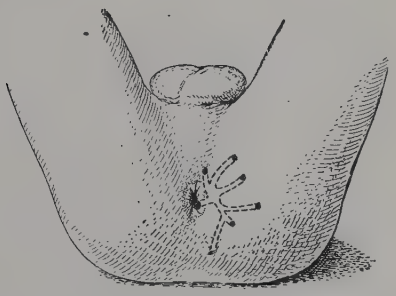
Diagnosis.—A considerable proportion of the patients have an idea of the nature of their trouble and not a few make the statement that he or she has a fistula. Careful inspection will at once disclose any external opening. As a rule in all cases they are found in the centers of elevated papillæ, though sometimes they occupy depressions, and occasionally in tubercular fistulæ are marked by considerable loss of substance. When the number and location of external openings are determined, palpation of the region should be engaged in to see if the finger can detect induration surrounding the sinuses, and it is often possible to trace out the entire course of a sinus by this method alone.

Diagnosis.—A considerable proportion of the patients have an in some cases will pass with but little effort readily along the tract into the bowel. This should be done without the finger introduced into the bowel because this is liable to deflect the probe. If it is not possible to pass the probe into the bowel, even after bending the point into various positions and trying in various directions, it must be remembered that usually there is only one internal opening in all fistulous cases, and that as a rule it is situated in the anal canal, generally within less than an inch of the anal orifice. Sometimes it is possible to find the internal opening by the injection of a colored fluid through the external opening and endeavoring to locate its point of escape into the bowel. For this purpose a solution of methylene blue or milk may be employed. If the case is coming under operation it is not important to persist with the various means of locating the internal opening, as this can be done much better under an anesthetic.

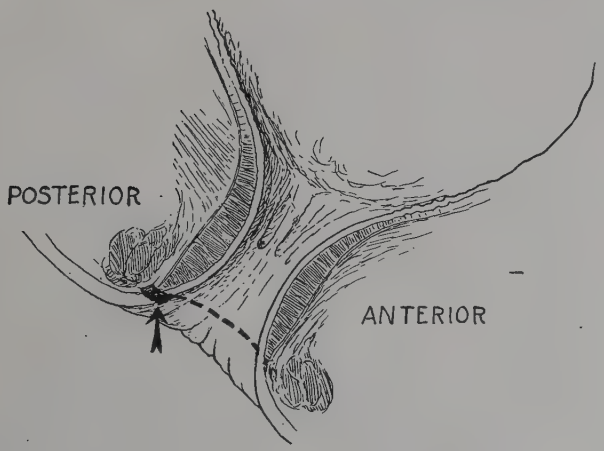
Subcutaneous.



Horseshoe.



With multiple external openings.



Usual site of internal openings.

Fig. 148*a*.—Types of anal fistula. (*Cooke.*)

Treatment.—While fistula is not concerned in the life of the victim, there is no doubt that the vitality is often lowered and the health undermined by the condition, and for this reason it is important that whenever symptoms denoting its presence bring the case under observation, operation should be advised and insisted upon. Treatment is divided into non-operative and operative.

Various measures of non-operative treatment have been advanced, mainly the application of irritant agents, such as a strong solution of nitrate of silver, or a 1 to 250 solution of formalin, or pure carbolic acid. The elastic ligature method was at one time popular but recently has fallen into disfavor. Lastly is the bismuth paste method of Beck's which is a 33⅓ per cent. mixture of subnitrate of bismuth in sterile vaseline. This is melted over a water bath, drawn into a syringe which accurately fits the external opening into which the paste is injected. This injection is made with sufficient force to insure the paste being carried into and completely filling all sinuses. It has been suggested that the curative action of the bismuth depends upon a gradual hydrolysis of the bismuth ingredient, with the slow liberation of nitric acid, which acts as an irritant to the tissues. It is probable that in about a third of all cases of fistula, Beck's paste if properly employed is curative. While the Beck's paste may be used and given a trial of a few weeks, particularly in cases where for whatever reason operation is not advisable, the best method of treatment is operation. There is no doubt that in the simple types of cases in which both the external and internal openings are readily found, the average surgeon is capable of radically curing these patients, but as a rule the man who is familiar with operating upon these cases is best able to render complete service. One not uncommonly sees cases that have been operated upon by general surgeons and still not cured. One of the reasons of this is that often the fistulous canals are not paid enough attention to in the technique of examination.

The methods advised are (1) fistulotomy, in which the entire length of the fistulous tract is excised. This operation has fallen into disfavor and rightfully should be abandoned; (2) simple incision, in which the sphincters are first well dilated and the interior of the rectum irrigated with an antiseptic solution. A grooved director is put through the sinus, and all the tissues cut through, from the external to the internal opening. The fistulous tract is then curetted or dissected out and the wound is packed with gauze sufficiently tight to prevent oozing, the dressing being completed with a T-bandage. A week or more in bed is required, the wound being packed particularly after the bowels have moved. Some of the proctologists advise the

application to the wound of a 15 to 25 per cent. solution of argyrol, applied by means of a small cotton swab, or a 10 per cent. mixture of balsam of Peru in castor or olive oil; (3) incision or excision, fistulectomy with immediate suture. In some cases this is the operation of choice, particularly when there are tortuous fistulous tracts. It is accomplished by having a probe in the tract, cutting down upon it and by careful dissection extended around all portions of the tract the tract is taken out *in toto*, suturing of the skin being required.

Perhaps no operation which may be termed minor in nature requires as much patience and skill as does that for the cure of blind (incomplete) internal fistulæ. Careful search must be made for the internal opening which is not always easy to find, and if found in the course of the sinus may not be easy to delineate. However, the sinus should be laid open and curetted before cure can be brought about.

Operations for the radical cure of rectovaginal or rectourethral fistulæ are found best described in books on general surgery or proctology.

ABSCESSES OF THE RECTUM AND ANUS.

Localized inflammation of the perirectal tissues is practically always the result of infection. It usually terminates in suppuration. The organisms found are the usual pathogenic types, tubercular abscesses being mixed infections with the streptococcus, staphylococcus, or Bacillus coli communis. Commonly the abscess is a pus accumulation beginning in an already formed fistulous tract, the abscess sac breaking externally, or when incised, completing the fistula. Abscesses about the rectum and anus occur frequently, these presenting perhaps 25 per cent. of the cases of anal affections. The levator ani muscles separate the rectum from the anus, and abscesses which occur in and above this point are known as supralevator, while those which occur below the level of the muscle are known as infralevator, the latter being divided into marginal or anal, intramural and ischiorectal. Those occurring in the neighborhood of the anus generally rupture on the outside or between the two sphincter muscles, while one occurring in the triangular space posteriorly to the anus, may rupture into the ischiorectal fossa or through the skin. Those above the levator ani muscle usually occupy the superior pelvirectal or the posterior pelvirectal space.

MARGINAL ABSCESSES.

These occur at the anal margin and involve only the superficial tissues of the skin or mucous membrane. They vary in size from a small point not much larger than an acne lesion to that of a pocket as large as a hazel nut. The abscess involvement of this area rarely involves the deeper tissues such as those in the ischiorectal fossa. They are usually single but may occur in pairs or larger numbers.

As a rule they are due to traumatism but they can be secondary to thrombotic hemorrhoids, infection of skin follicles, etc. Pain is the most prominent symptom of marginal abscesses, which might be quite severe even though the lesion be small. Tenderness is extreme, and as a rule there is no constitutional disturbance.

The treatment of marginal abscesses is surgical, although generally after poulticing with hot applications of some sort they rupture. The attendant distress is so severe that it is wiser under local anesthesia, such as ethyl chloride, to incise the abscess cavity and gently express the pus, insert a small piece of well-oiled gauze between the edges of the incision at the first dressing, and in a few days the condition is over.

Subtegumentary or perineal abscess occurs beneath the skin of the perineum or between it and the mucous membrane of the rectum. It is practically always secondary to some other disturbance, and results from infection of the lymphatics. It occurs most frequently in hearty, robust men, and seldom in women or old people. Poor technique in the injection of hemorrhoids with carbolic acid is a frequent cause of this form of abscess. The symptoms of this form of abscess vary. As a rule there is no distress, and the diagnosis is often not made until a discharge of pus from the rectum occurs. A sense of dull aching and discomfort in the part, and in some cases moderate constitutional symptoms, discomfort in sitting or walking or rubbing of the clothes is often complained of. When the abscess develops above the sphincter it may open into the lumen of the rectum and then gives no external evidence. However digital examination would then have disclosed a globular mass indurated or fluctuating according to the phase of its development.

Treatment.—The natural course of these abscesses is to rupture spontaneously. If the abscess ruptures through the skin the result is a blind external fistula. If it opens within the rectum or anal canal it is a blind internal fistula, or if through the skin and also into the rectum it forms a complete fistula. The distinguishing feature about these abscesses is that they do not drain and heal like abscesses elsewhere but continue to remain as sinuses.

If seen early they may be aborted by painting with tincture of iodine or by Bier's hyperemia treatment. As a rule, they are well developed, in which instance they should be opened by a crucial incision and swabbed out with a little carbolic acid and alcohol. The cavity should be thoroughly flushed out at least once a day with physiologic salt solution or mild antiseptic. Rest in bed is essential to rapid recovery. If the abscess occurs posteriorly, incision may be made from the anus to the coccyx and carried through the external sphincter muscle. As a rule packing lightly with iodoform gauze to establish drainage, or the use of a rubber tube or cigarette drain is required for the first twenty-four hours, after which the abscess cavity is dressed every two or three days until it is thoroughly healed. Lynch states that when these abscesses are under the sphincter but do not rupture into the anus it is seldom necessary to cut the sphincter.

ISCHIORECTAL ABSCESSES.

These represent about 18 per cent. of the rectal abscesses. They develop outside of the rectum and beneath the skin and fascia. They may be single or multiple and when multiple may and generally do connect behind the rectum through the space between the levator ani and the sphincter. The cavities in which these abscesses develop are filled with fat, and as the pus fills the spaces, the fat is displaced. The connective tissue remaining gives the abscess a honeycombed form instead of being one large abscess. There seems to be no limit to where these abscesses may travel. Frequently an abscess occurring on one side will burrow to the opposite, perhaps behind the rectum and form a horseshoe or a bell shaped cavity. These occur more frequently in men than in women, and in the majority of instances are due to some lesion of the rectum or anal canal. Not infrequently, even though operated upon they may recur years afterward, showing that the infected material has a considerable degree of virulence; although probably the sacculation incident to the trabecula of connective tissue found in the ischiorectal fossa, and the fact that some tracts are not opened and drained, are responsible for recurrence.

Symptoms.—As a rule ischiorectal abscesses develop acutely with constitutional reaction such as a slight chill followed by headache with temperature from 104° to 105° F. Locally there is a vague feeling of soreness within the rectum which gradually increases to a dull ache, and later a throbbing pain which increases on defecation. After a while the patient becomes incapacitated and the slightest movement brings on the most intense suffering which continues until the abscess ruptures or is opened with a knife. The location of the abscess some-

what determines the severity of the pain. If this is situated near the anus where there is either considerable loose or areolar tissue they are less painful than those situated where the sphincters are firmly bound by muscle and fascia. If the cavity is close to the sphincter there is considerable spasm which increases the pain.

A finger introduced into the rectum and pressed up and down will usually feel a circumscribed mass of induration or fluctuation. Bimanual palpation with one finger in the rectum and counter pressure on the perineum is of much value in early diagnosis.

Treatment.—The cure of ischiorectal abscess depends wholly and absolutely upon the plan of treatment adopted. While poulticing and allowing it to take its own course may mean its rupture externally, it

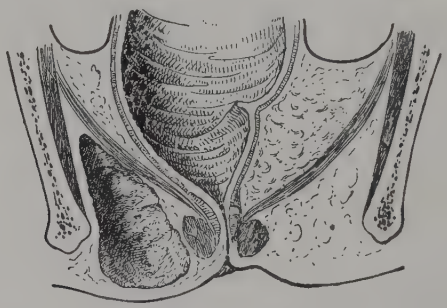


Fig. 149.—Ischiorectal abscess. (*Cooke.*)

is more liable to be followed by fistula than if free incision is made. Thus it is wise not to wait for fluctuation unless the individual is syphilitic.

The diagnosis being made, the abscess should be opened by a longitudinal incision. All its trabeculæ which cause pocketing of the pus should be broken down and the cavity packed with iodoform gauze. A drainage tube should be inserted and the gauze packed loosely around it. This tube with gauze can be removed after the first twenty-four hours, and packing engaged in until the wound is healed up from the bottom. It is a good plan not to pack these abscess cavities too tightly, just enough to keep the walls apart so that pocketing does not occur. The reason for this is that in tightly packed abscess cavities the formation of fistulæ is encouraged. Curetting is not advised because the steel-spoon affords no knowledge of the condition of the walls, while the educated finger distinguishes necrotic from normal connective tissues. The operation is best done under a general anesthesia, nitrous oxide answering the purpose.

If it is assumed that abscess is tubercular in character the cavity may be swabbed out with tincture of iodine or pure carbolic acid followed by alcohol, after which it should be treated in the ordinary way.

General sepsis may follow or accompany an ischiorectal abscess, but it is a rather rare occurrence. Its most frequent sequelæ are external rupture, internal rupture, or involvement of the opposite fossa with consequent horseshoe fistula.

RETRORECTAL OR SUPERIOR PELVIRECTAL ABSCESSES.

These are supralevator in location and may result from necrosis of the pelvic bone or from perforation of the rectum by a foreign body, or from prostatic or seminal vesicle infection. If they are allowed to continue their course considerable destruction of tissue may result, the abscess rupturing into the peritoneal cavity with fatal termination or it may open into the inguinal region anteriorly, resulting in a chronic fistula, or gravitate into the posterior pelvirectal space.

Symptoms.—The symptoms at first are vague and indistinct. Generally there is a dull pain or sacral ache and a heaviness in the pelvis or sciatic region. There may or may not be pain on defecation. Generally there are signs of pus formation—namely, temperature, leucocytosis and sallow complexion. External palpation about the anus and perineum may elicit no sign. Digital examination within the rectum, however, demonstrates a circumscribed induration back of the rectum, or later, as pus forms and the tension increases, the mass becomes painful, fluctuates and obstructs defecation. Retention of urine may occur.

Treatment.—The proper method of opening these abscesses is important if the patient is to get the best results. This is done in the lithotomy position, the incision being made according to the point of bogginess. Usually it is required that the incision goes deep enough, somewhat through the levator ani muscles so as to split this muscle and thus the wound does not draw together, and thorough drainage be interfered with. As a rule dressing with a drainage tube with gauze lightly packed around it are required. Lynch advises that if a fistula results it can be treated by injections of Beck's paste. If the abscess is situated in the urethrorectal space it is best reached by a posterior incision between the anus and the coccyx; if in an anterolateral space, a similar incision between the anus and the sacrum, followed by careful dissection until the pus is reached. In the latter in the female the vaginal route is available.

PRURITUS ANI.

Pruritus ani is an exceedingly common and distressing condition concerning which much has been written, particularly on the discussion of etiology. A perusal of the literature shows that there have been numerous beliefs as to the cause of this condition. From a functional cutaneous affection, through all sorts of neurological classifications, even from some metabolic disturbance, numerous have been the theories advocated. There is no doubt, however, that pruritus ani is due to an irritation of the anal or perianal skin, the etiology of which most probably is that advocated by Murray,[2] namely that it is due to a Streptococcus fecalis infection of the deeper skin level. This conclusion of Murray's, and I have confirmed his findings, was arrived at by finding the Streptococcus fecalis in the substance of the skin, this not being true in normal conditions.

There is no doubt that in abnormal conditions about the anus such as simple fissure, ulcer, particularly in the posterior median line between the two sphincters, diseased crypts, papillitis, and other conditions such as diabetes, etc. may cause an itching in the skin at this location. The itching may also involve the skin of the scrotum or vulva. Instances of proctitis, particularly the hypertrophic form due to saccharo-butyric intestinal toxemia, causes more or less leaking of the mucosa of the rectum and lower colon, a moisture making its exit onto the anus brings about changes in the skin, a loss of resistance, perhaps a Streptococcus fecalis infection, or even if not, more or less itching until the cause of the discharge of the mucus has ceased. For the same catarrhal reason thread worms may cause more or less itching due to a catarrhal condition of the mucous membrane which may be set up particularly in young persons.

In the array of other causes certain constitutional and reflex conditions have been mentioned. Those of constitutional origin, other than diabetes mellitus, cannot stand close analysis and scientific investigation. The same may be said of the reflex causes. When such conditions as urethral stricture, hypertrophied prostate, seminal vesiculitis and vaginitis are mentioned as reflex causes of pruritus ani, one has only to stop a moment and think of the many cases of these conditions, including discharging conditions of the vulva, vagina and uterus in which pruritus is not present. Liability to infection is characteristic of diabetes mellitus, and while the itching may be due to local irritation from the glucose, not a few diabetics with pruritus have Streptococcus fecalis infection of the anal skin.

It used to be taught quite properly that there were three causes for hay fever, some local condition in the nose, a neurosis, and the pollen of plants, these three being necessary for the development of a typical case. In my opinion the same rule holds true in pruritus ani —namely, a local cause in the rectum and anus, such as has been mentioned, a neurosis, and an exciting or precipitating cause, namely

Fig. 150.—Pruritus ani. (*Cooke.*)

an infection of the skin of the Streptococcus fecalis. The first of these may be absent, the second might be somewhat of a limited factor, but is always more or less present, while the third is a finding in all cases of true pruritus ani and in most of those of the posterior scrotum and vulva.

Symptoms and Diagnosis.—A patient comes to the physician complaining of an itching in the anus or in the perianal region. The itching is usually worse at night after the patient has retired to the warmth of his bed, and in most instances the itching is of a character

that is not relieved by scratching—in fact the more traumatism from the scratching, generally the worse the itching. In the beginning there are no changes in the skin to mark the presence of a pruritus. In some cases, particularly those of long standing, the damage inflicted by the patient in scratching causes more or less changes in the skin. However the majority of cases do not show any peculiar local condition, and such as may be present is overlooked, and may consist merely of a narrow zone of thickened integument encircling the anus, dead white in color from the loss of natural pigment. In some cases this area is extensive, going out far enough to involve the perineum and scrotum or vulva anteriorly. In not a few of the instances the itching is so intractable and difficult of control that because of loss of sleep the general health breaks down and not a few of such individuals have taken to the use of narcotic drugs, some have committed suicide, and neurasthenia, melancholia and even insanity have been known to result.

The diagnosis can always be made by the patient alone. Examination of the integument about the anus may or may not serve to confirmatory purpose.

Treatment.—All cases of pruritus ani should be given a thorough local and general examination. Such lesions as may be found in the rectum or anus would require attention, and in instances of proctitis due to saccharo-butyric intestinal toxemia, proper dieting and care for that condition is essential. In the pruritus vulva cases careful examination should be made of the vagina and uterus and the sources of discharge stopped or controlled by douching.

When these have been attended to, or when no local condition is found to be treated, the parts should be kept scrupulously clean. Advice should be given that after the bowels have moved the anus should be wiped by toilet paper from before backward and not carry the possibility of infection forward into the perineal, scrotum or vulva integuments. It is best after the patient's bowels have moved that the parts be cleansed with a soft cloth, preferably gauze, using a 5 per cent. salt solution or a 20 per cent. solution of witch hazel, slight bearing down being engaged in during the last part of the cleansing so that the skin within the anus and anal folds will be well cleansed.

If the skin is dry and brittle, an ointment of oleate of mercury smeared on a piece of cotton and the cotton applied to the part, the ointment being removed occasionally with a 50 per cent. solution of alcohol, answers to good purpose. Occasionally when the skin is moist and boggy two or three exposures to the X-rays will sometimes bring about marked benefit, with a cure in a small proportion of cases. The

main reason why a cure has been brought about by the X-rays in these cases is that the effect of the X-rays is to cause a thinning of the skin, by destroying the local circulation and causing an atrophy of the skin cells, this usually being accomplished by atrophy of the secreting glands of the skin, and a dry, wrinkled, parchment-like result is accomplished. By bringing about this atony of the skin any infection that there may be in the corial layer is deprived of its nutrition and its means of sustenance is destroyed, with perhaps a cure of the itching. With some of the present X-ray methods the dosage is so small that no skin change is accomplished, possibly only an erythema; or if large enough or long enough kept up doses are given, a burn which intensifies the condition finally, although perhaps relieving the distress for the time being. In the old days when a soft tube energized by static machine or with small coil was used better results were accomplished by the X-ray treatment of pruritus ani than is brought about today by the use of the transformer currents and the Coolidge tube. Many cases are met with in which no amount of X-raying of the skin seems to bring about an atony of the anal region and if so no benefit to the pruritus, and there are other cases in which it is unwise to keep up the amount of exposures necessary. The benefits from X-ray treatment are always questionable but may be tried.

Various local applications have been advised for the itching. Among these may be mentioned carbolic acid in lotion or ointment, the strength varying from 1 to 5 per cent., a 2 to 4 per cent. menthol or chloral, a 2 to 10 per cent. cocaine oleate or what answers to good purpose in my hands, a silver ointment (unguentum Credé). Daily painting of the parts with tincture of benzoin answers a good purpose in some cases. Various soothing applications have been used such as Goulard's cerate or lead acetate ointment, freshly made, belladonna ointment, calomel ointment 4 per cent., oxide of zinc ointment, etc. Some authors have advised stimulating applications—such as the use of oil of cade, (1 to 2 per cent.), balsam of Peru (10 per cent.), compound iodine ointment, ammoniated mercury ointment, ichthyol ointment (10 to 20 per cent.), a painting of the parts with a solution of nitrate of silver (2 to 4 per cent.), this being done daily. Others again resort to caustic applications such as Churchill's tincture of iodine, pure carbolic acid, the saturated solution or solid stick of nitrate of silver.

Dusting powders as talcum and stearate of zinc, camphor (2 per cent.), calomel (5 to 10 per cent.), menthol (1 to 2 per cent.), salicylic acid (1 to 2 per cent.), carbolic acid (1 per cent.), and others have been used.

Of the various prescriptions advised the following is offered:

℞ Ichthyol	ʒss	2.0
Zinc oxide, Lanolin	āā ʒij	8.0
Oleum olivæ, Aqua calcis	āā ʒss	15.0

Sig.: Apply night and morning.

Cooke advises:

℞ Ext. hamamelis fl.	ʒj	30.0
Ext. ergotæ fl., Ext. hydrastis fl., Tr. benzoin. comp.	āā ʒij	8.0
Ol. olivæ vel Ol. lini (carbolized 5 per cent.)	ʒj	30.0

M.

Sig.: Inject 2 drams into the rectum at bedtime.

℞ Calamine	gr. xl	3.5
Zinc oxide	ʒiij	12.0
Liquor carbonis detergens	gr. xx	1.4
Glycerin	ʒss	15.0
Aqua calcis	ʒj	4.0
Aqua	q. s. ad ʒiv	120.0

M.

Sig.: Apply twice daily.

℞ Carbolic acid	ʒij	8.0
Salicylic acid	ʒj	4.0
Glycerin	ʒj	30.0

M.

Sig.: Apply to the parts with a camels' hair brush when itching is severe.

℞ Zinc oxide	5 grams	0.3
Iodine	25 grams	1.6
Glycerin	50 grams	3.3

M.

Equal parts of a solution of argyrol and ichthyol will occasionally be found useful.

Other formulas are:

℞ Calomel	ʒj	4.0
Carbolic acid	gr. iij	0.2
Tar	ʒss	2.0
Menthol	gr. xx	1.3
Zinc oxide	ʒij	8.0
Lanolin	q. s. ad ʒij	60.0

M.

Matthews recommends:

℞ Campho-phenique,
Distilled water .....āā ℥j | 4.0

This should be applied as a lotion after the use of hot water, and repeating as often as necessary. Adler suggests the following as worthy of trial:

℞ Fluidextract of hamamelis ..... ℥j | 30.0
Fluidextract of ergot ..... ℥ij | 8.0
Fluidextract of hydrastis ..... ℥j | 4.0
Compound tincture of benzoin ..... ℥ij | 8.0
Carbolized olive or linseed oil,
Carbolic acid (5 per cent.) .....āā ℥j | 30.0
M.
Sig.: Shake well before using.

Allingham recommends the following:

℞ Menthol ..... ℥j | 4.0
Cocain hydrochloride ..... gr. xx | 1.3
Alcohol,
Distilled water .....āā ℥j | 30.0
M.
Sig.: Apply locally.

℞ Liquor carbonis detergens,
Wright's glycerin .....āā ℥j | 30.0
M.
Sig.: Apply locally.

The parts are to be painted with this once or twice a day. If the perianal tissues are very much thickened, the following will be found beneficial:

℞ Liquor potassæ,
Cade oil,
Alcohol .....āā ℥j | 30.0
M.

This is rubbed into the parts once a day, and followed by a soothing ointment, such as:

℞ Unguentum zinci oxidi,
Chloroform .....āā ℥j | 30.0
M.
Sig.: Apply freely to the parts, but allow the chloroform to evaporate before covering.

While the above measures answer to sufficient purpose in the minor cases, in my experience they failed in most of those which I have seen. It is true that even in the severe cases where possibly after an operation upon hemorrhoids, cryptitis, or some local condition, they answer to sufficient purpose, most of the cases do not present an anatomical lesion, and not a few have an anatomical lesion removal of which does not do away with the itching. Thus the above measures may be employed but often when persisted in failure is the result. For that reason Dr. Murray's work interested me to the extent of making studies of all the cases I have seen recently in which his etiological belief could be substantiated and I am satisfied that he is correct in the vast majority. While many have criticized his work and have not had results from the use of vaccine, the more experience I have in persistent cases, the more I am convinced that Murray has advanced the best means of medical treatment of this distressing condition, and a treatment which is often of more value than local applications and even surgery. When an infection occurs, the resistance of the parts is lowered and in that way the infection is kept up, naturally because the opsonic index of the individual against the Streptococcus fecalis is low. Since pruritus ani rarely extends above the white line of Hilton it is distinctly a cutaneous affair, and I believe with Murray that in some cases the skin moisture produced locally is due to the lowered condition of the infected skin. He contends that pruritus ani, vulvæ or scroti are not a part of a diabetic condition and that in diabetes resistance to infection is lowered making possible a Streptococcus fecalis infection of the skin to account for many of these also.

The isolation of the Streptococcus fecalis, according to Murray, is accomplished as follows: "The swabs received are gently but firmly rubbed over the surface of solidified standard beef infusion agar in Petri dishes. The agar should have an acidity of 0.5 to phenolphthalein. The small clear colonies of Streptococcus fecalis are readily distinguished from the larger colonies of Bacillus coli, Staphylococcus albus and other bacteria encountered in such cultures. The small colonies of Streptococcus fecalis are sub-cultured on slants of the same kind of agar as that used for isolation. As a rule, the growth is rapid and luxuriant and a few tubes suffice for the amount of bacterial vaccine desired. However, some strains are encountered that are grown with difficulty. The bacteria should be killed either by 0.5 per cent. phenol (carbolic acid) solution or a 0.33½ per cent. tricresol solution. Heat should not be used.

"After the vaccine is proved sterile, it is ready for use and should be kept in a sealed bottle in a cool place. At the time of using, the

vaccine should be thoroughly shaken in order that the dead bacteria may be evenly distributed in the dose given. The dosage is begun with 3 minims, injected into the buttock close under the skin, not into the muscle. The first three or four doses are repeated at intervals of two days, increasing from 3 to 5 minims at each treatment, unless a severe reaction is caused by the previous dose. The number of minims at each treatment may then be increased as much as the judgment of the operator dictates. The results will be obtained in most of the cases just as well without local applications of anti-pruritics except for the temporary relief obtained."

After considerable experience I would criticize the unfavorable reports which have appeared regarding the use of this method of treatment along four lines. (1) The technique of getting the swabs for the bacteriological work and isolation and the Streptococcus fecalis not being obtained; (2) That large enough doses of vaccine were not utilized, because in some of my cases doses of a billion, and even nearly two billion have been necessary toward the end of the treatment—in fact one case which started at 75 million accomplishing a reaction, in the fourth month was given four billion and a quarter, and even then the reaction was minimal; (3) That long enough time was not persisted in, in the use of the vaccine; and (4), and this is important, that stock vaccine was used instead of the autogenous.

Various surgical methods of treatment have been devised. Since the vaccines, however, I doubt if they are indicated any longer. These may be divided into two classes—those which have for their object the destruction or removal of the altered skin and those in which the severing of the nerves supplying the infected parts is undertaken.

Cauterization of the area involved with the actual cautery has been very popular with American surgeons and has been beneficial in some cases. In addition to this various operations for the removal of the altered skin by radical dissection has been suggested although this is a procedure that is practically never warranted.

The second class of surgical procedures consists of those which are based upon the idea of disposing of the pathologic sensation by severing the nerves which transmit it. The first of these was devised by Ball[3] in which a curved incision is made on each side of the infected area enclosing the entire ellipse with the exception of a narrow neck in front and behind, these incisions being carried down to the sphincter muscle. Careful dissection with scissors on the surface of the muscle around its anal margin and up the anal canal to above the mucocutaneous junction is performed, the dissection extending around

the entire circumference, all connections with subjacent tissues being divided. The adjoining areas in front and behind are undercut to a point well beyond the area of irritation, the flaps are firmly replaced and retained by sutures, with a few intervals left between them for drainage.

The second method, which has far more to recommend it than the above, is that of Lynch.[4] This can be done under local anesthesia such as a 1 per cent. solution of novocaine, or a ⅕ per cent. solution of cocaine, or ½ per cent. solution of urea and quinine hydrochloride. With the patient upon his left side, the knees and thighs flexed, a point is chosen about one and one-quarter inches from the anus transversely on each side. Injection is made under the skin to a point posterior to the anus and a point anterior to the anus almost meeting in the median line. The area surrounding the posterior midline is anesthetized. At the point above mentioned a small curved incision is made about a half inch long and extending just through the skin. In this incision a blunt-pointed dissecting scissors curved on the flat is introduced and a blunt subcutaneous dissection is carried out to the anus mesially and to the raphés anteriorly and posteriorly. When completed, there is an area of skin extending from the anterior raphé to the posterior commissure and involving all the skin within a radius of one and a half inches from the anus, which has been deprived of its sensory nerves. Generally the wound heals quickly and the itching of course ceases immediately. What is important to remember, even with this operation, is that though the nerve fibers be cut, in the course of time they regenerate and sometimes the condition of itching re-establishes itself as before. This takes from six months to two years.

CRYPTITIS AND PAPILLITIS.

The anal mucous membrane is thrown into a number of longitudinal folds, to allow for dilatation of the anus without undue stretching of the mucous membrane. Between each fold is a depression which dips down and terminates in a little pocket. These pockets are known as the crypts of Morgagni, and form a sort of semilunar valve between each column. Between each of these valves is a little tactile body known as a papilla. These papillæ may enlarge, which is most commonly seen in hemorrhoidal conditions, and may reach relatively enormous proportions.

CRYPTITIS.

Pain directly at the anal orifice is the most reliable symptom of cryptitis. It is usually dull and aching in character or it may be sharp and throbbing in nature if an abscess forms. The pain is increased during defecation, made worse by exercise and aggravated on standing. It is sometimes considerably relieved by pressure. Sometimes in

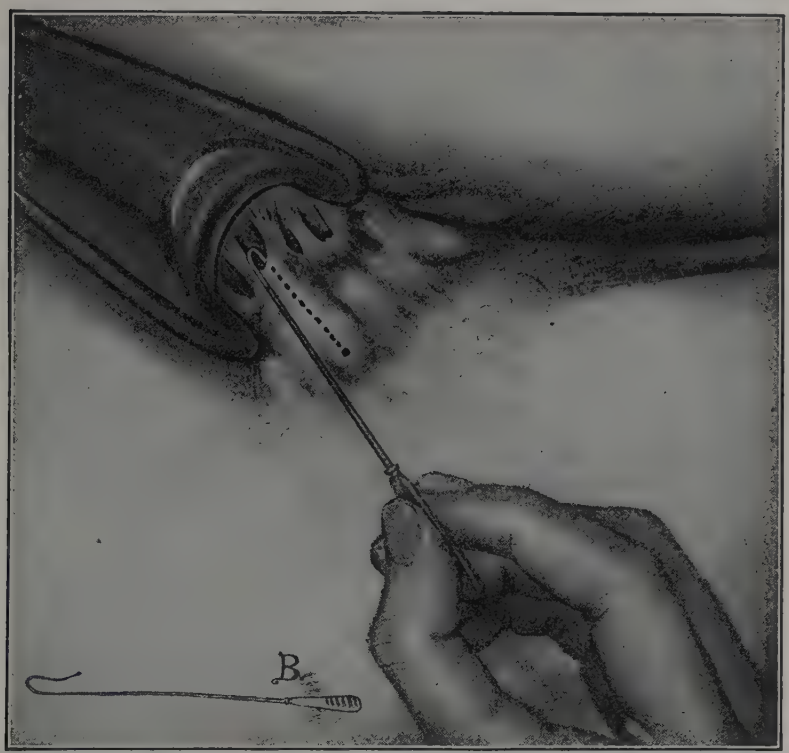


Fig. 151.—Exposure of the anal canal through Humphrey's speculum; shepherd's crook probe introduced into cryptic pocket leading down into hypertrophied radial fold. *B*, Shepherd's crook flexible probe. (*Tuttle*).

cryptitis there is painful urination, on account of spasm of the neck of the bladder. Other reflex disturbances are dysmenorrhea, amenorrhea, dysuria, spasm of the sphincter or levator ani, and neuralgia of the testicles and perineum.

The diagnosis is made from the symptoms and by directly investigating the condition of the crypts. This is best done by the use of a Humphrey's speculum, or the use of any speculum with a window on one side, with a probe bent upon itself, inserted into the rectum

and each crypt gently investigated. The one which is inflamed causes severe pain the moment the probe is inserted.

Treatment.—If cryptitis is recognized in the early stages it is possible to cure the condition without operation. The crypt is exposed by the use of a fenestrated rectum speculum and the bent probe dipped in pure ichthyol and passed through the speculum until the crypt is reached. The ichthyol is then carried into the crypt and in this way is applied directly to the ulcerated area. This process is continued daily until all pain has disappeared.

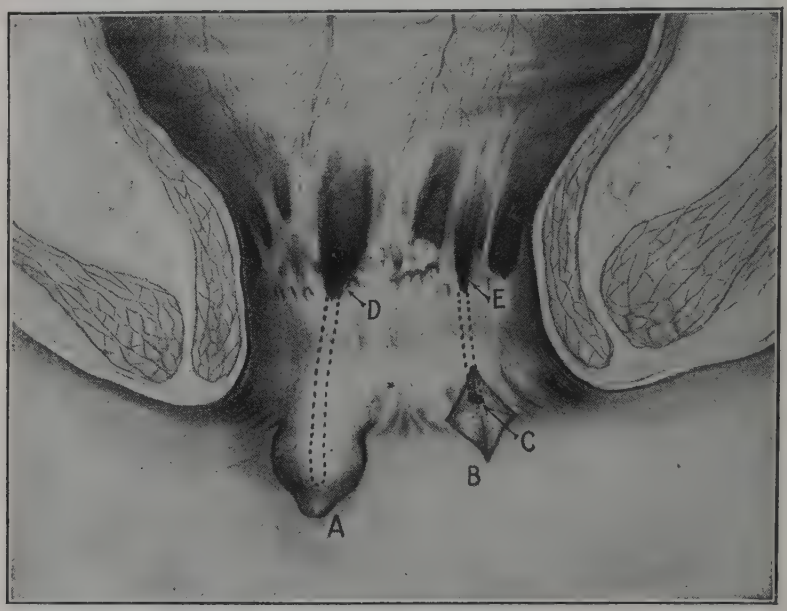


Fig. 152.—*A-D*, Cryptic tract running down into skin tabs. *B*, Wound from which skin tab has been clipped off. *C-E*, Part of cryptic tract unopened by clipping off skin tabs, thus leaving a subcutaneous fistula which constantly infects the wound from which the skin tab has been taken off, and thus prevents the healing. (*Tuttle.*)

If this is not successful, operation is required. This is done under local anesthesia. The crypt is infiltrated with cocaine, the bent probe is passed into it, the valve put on tension, and when the valve is removed by a scissors curved on the flat. The daily application of some healing ointment is required.

PAPILLITIS.

Inflammation of the papilla or papillæ, may be independent of a cryptitis, but generally is associated with it. Not uncommonly it is

associated with proctitis or simple congestion of the anus; also with hemorrhoids.

Symptoms.—The most constant symptom of papillitis is a tickling sensation at the anus which often leads to the diagnosis of worms instead.

Treatment.—The treatment is surgical, in which the papillæ are removed under local anesthesia. The papillæ are directly anesthetised at the base, brought into view with artery forceps, tied with a silk thread, and cut off or allowed to slough away.

PROLAPSE OF THE RECTUM.

Prolapse of the rectum may mean either a slight invagination of the mucosa, a complete sagging of all the walls of the bowel, or the invagination or intussusception of one portion of the bowel into the other. It is divided into incomplete and complete. By incomplete is meant that form of prolapse in which the mucous membrane only is involved. Complete prolapse or procidentia is where all the tunics of the bowels are concerned.

PARTIAL PROLAPSE.

This is the more common variety of the two and a whole or any portion of the circumference of the gut may be involved. When the trouble occurs independently of the dragging action of polypi or internal hemorrhoids, the entire bowel is apt to present a more or less uniform involvement. The condition is not uncommon in childern and may be met with in very young infants. The aged in whom absorption of the fat and relaxation of the parts has occurred are also frequent victims.

The causes of partial or incomplete prolapse are age, such as the young in which the curves of the sacrum are much less developed than in adults and in consequence the expulsive force of defecation is applied more nearly in a straight line with the outlet of the bowel; the old, in which it is due to defective muscular tonicity, a condition of the sphincters, prolonged constitutional disease which results in enfeeblement and emaciation, and spinal disorders resulting in an interference of the nerve supply of the parts or faulty operation in which the sphincter muscles are permanently injured, or the presence of polypi or internal hemorrhoids which may act mechanically in producing diversion of the mucosa. The most important or exciting cause is straining at stool. This condition, when it has occurred, and even though it is reduced properly, is very liable to occur again. The more times it

occurs the more relaxed the parts and greater the liability of its continuation.

Symptoms.—The onset of incomplete prolapse is probably always gradual and therefore has no appreciable symptom. The condition continuing there comes on a sensation of discomfort, a feeling of fullness and perhaps a little burning and irritation about the anus. There soon follows a profuse catarrhal discharge, often blood-stained, and perhaps an annoying pruritus. Visual inspection is all that is required for diagnosis.

Treatment.—The first indication is a reduction of the prolapse, which is accomplished by anointing the part with a lubricant and its slow careful return within the anus. This should be done gently, remembering that the most dependent part of the prolapsed gut is usually from the highest section of the rectum.

Under palliative treatment, prevention of straining at stool is important, and for this reason the use of laxatives or liquid petrolatum are required. It may be necessary to use some form of truss to support the parts and prevent protrusion. For this purpose defecation in the recumbent position, and then a compress applied to the anus or buttocks which are strapped together with adhesive plaster before the upright position is assumed serves to good purpose. In the adult a surgical operation alone gives satisfactory results. Of course the general health should be taken care of, the body strength built up, and all that pertains to reconstruction of the general body engaged in. In tabes operation is out of the question.

The procedure used is the method of linear cauterization devised by Van Buren and consists of drawing lines with the point of the actual cautery heated to a dull red at several different sites of the mucous membrane, beginning above the prolapse and extending them downward parallel with each other onto the skin of the anus. In this way cicatrices are formed which bind the mucosa firmly to the muscular coat, and at the same time the patulous anus is narrowed. Only four lines of cicatrization will be required provided they are made sufficiently deep, and complete healing may be expected in two weeks. If the causes underlying the condition have been previously removed, permanent cure may be expected. Other methods of treating prolapsus are those described for the treatment of internal hemorrhoids.

COMPLETE PROLAPSUS.

When all the coats of the bowel descend from their normal relation the condition is one of complete prolapsus. This is found far more frequently in adults than the incomplete variety, and men are more

often affected than women. The complete prolapsus naturally is much larger than the incomplete variety, and occasionally prolapses so as to project six or more inches from the anus and be as large as a fetal

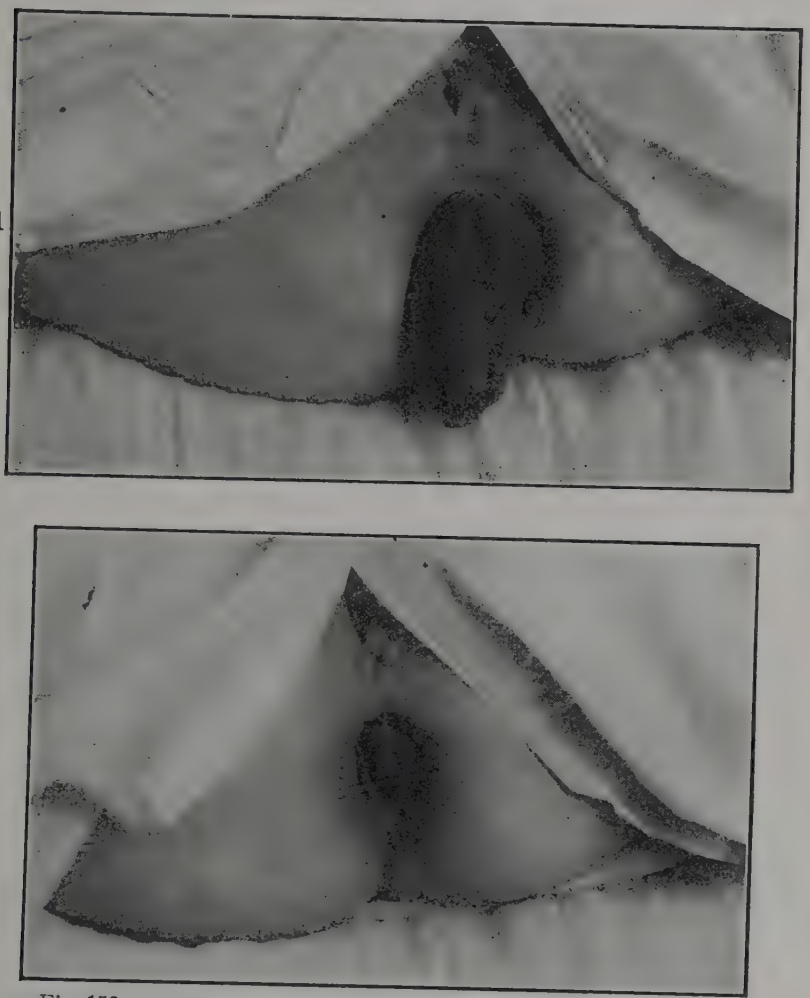


Fig. 153.—*A*, Prolapse, second degree, 17 inches long. Absolute absence of muscular and fascial support. *B*, Same case, showing relaxed or absent sphincters after prolapse is replaced. (*Martin.*)

head in circumference. The etiology and symptoms are the same as in the incomplete variety.

Treatment.—There is no question that women who have borne children have more or less laxity of the rectal mucosa and therefore such conditions as sub-involution of the uterus, rupture of the perin-

eum, levator ani muscles, relaxation of the broad ligaments, and impaired displacement of the uterus should receive attention. The inveterate use of saline cathartics bringing about congestion and straining is somewhat of a factor; the same may be said of polypi, hemorrhoids, and other tracting rectal conditions.

The reduction of a complete prolapsus calls for skill and resourcefulness as well as the exercise of good judgment. The problem presented is usually far from a simple one. When the bowel is allowed to remain in a prolapsed position it rapidly becomes swollen and edematous, and in a short time may become agglutinated by the plastic material poured out, the entire tumor mass becoming firm and resistant like the tumor of an intussusception. When the case is seen early reduction by pressure should be attempted. The protruding mass should be lubricated with bland oil and after covering it with a soft piece of gauze attempts should be made to reduce its size by gently kneading and compressing it. The lower part of the tumor which is the highest portion of the gut should be reduced first. This should be done in the knee-chest position.

When it becomes apparent after reasonable perseverance that reduction cannot be effected by the above plan, it is better to administer a general anesthetic and divulse or incise the sphincter and replace the parts at once. In this case it will be wise to pack the rectum with oil gauze and adjust a firm compress in order to prevent recurrence during the ensuing time of nausea and relaxation. After reduction has been effected, the most important consideration is to prevent a recurrence of the trouble. To this end the feces should be rendered soluble, straining at stool carefully corrected, the rectum should be sprayed with a 1 per cent. solution of silver nitrate, the bowels should be evacuated in the recumbent position, and adhesive straps should be put across the buttocks so as to hold the two cheeks tightly together.

If when the case is seen it is decided that strangulation has taken place, and the viability of the gut has been so altered that its removal is advisable, rectorrhaphy, rectopexy, sigmoidopexy or resection or amputation of the prolapsus is advised. These operations are all on the order of major surgical procedures and are found best described in books on surgery of rectum and anus. With the exception of resection, my opinion is in the majority of instances that the complete operation requires a narrowing of the anal outlet, this being done by the method described by Dupuytren, or that of Lange which contemplates the narrowing of the lower end of the rectum as well as the anal canal. Van Buren's method of linear cauterization serves well in quite a few of the cases, as in those of the incomplete form.

FIBROUS STRICTURE OF THE RECTUM.

This is due to fibrous deposits in the form of annular, tubular, linear and valvular strictures. The annular stricture is a symmetrical occlusion involving the entire circuit in a certain plane of the rectum or anal canal. Tubular stricture is a term used to describe the form in which the exudated bowel extends beyond the limits so that some length of stenosis is present. Valvular stricture is due to one or more of Houston's valves being hypertrophied by virtue of hyperemia, plastic exudate or a fibrosis. Linear stricture, which is the most obscure and difficult to diagnose, is a scar-tissue segment of the bowel usually due to a healed ulcer or from operative interference for hemorrhoids, fissure or fistula. It represents a fibrous thickening in an area of the bowel wall. The picture may occur above the levator ani muscles, due to extension of malignancy in the pelvic cavity through the lymphatics.

The etiology of stricture is that of inflammation which has repaired—an inflammation which begins in the mucosa and extends to the submucosa and muscular structure infiltrating these tissues and leading to fibrous stricture formation. All of the cases have their origin in some previous intra-rectal injury, irritation or ulceration. As causes of inflammation, muscular spasm, dysentery, syphilis, gonorrhea, tuberculosis, traumatism, and various surgical procedures in the rectum are all factors of importance. The most important cause of anal stricture is the result of the so-called Whitehead operation.

Symptoms and Diagnosis.—The symptoms of fibrous stenosis of the anal canal or rectum are rather obscure and confusing in the early stages. In the ulcerative stage, local and reflex phenomena lead one to expect inflammatory change in the terminal canal. There may be present such symptoms as heaviness, dragging sensations in the loin or perineum, aching down one or both legs, especially if the lesion be in the anal canal, vague pelvic pains, or pain in the uterus or adnexa, and these may be mistaken for cystitis or proctitis owing to the proximity of the contiguous inflammation. Such signs as perineal moisture, perhaps a pruritus, frequent mucus discharges, tinged with blood and purulent material and pus, and blood and mucus with the stools, which are liable to be soft, are all characteristic ulcer symptoms.

In the period of obstruction, the symptoms may be quite vague and entirely those simulating constipation. Ribbon shaped feces may occur or the stool may have a caliber not any thicker than a pencil. Sometimes when the stricture is high, feces collects in the lower rectum, shape up somewhat, and the stool be fairly normal in caliber. As a rule, considerable accumulation of feces and gas will occur above

the stricture, which if severe enough will cause distention of the abdomen, with perhaps an irritable stomach, nausea and certain general symptoms resulting from auto-intoxication. In some cases periods of diarrhea will alternate with constipation, and this frequently happens in the form where more or less ulceration or abscess formation is present. If the stricture is low in the anal canal more or less tenesmus with a feeling of incomplete evacuation after movement are present.

The diagnosis is made by proctoscopic examination or by examination with the finger. By the use of the proctoscope the texture of the stricture, possibility of ulceration, color, resiliency and degree of obstruction are best noted. Under no circumstances should constriction be forced through, and if any effort is made in this way, it is better that it be done with the finger. Most strictures are in the lower four inches of the rectum and can be felt readily. In the female it is sometimes possible to feel well above the stricture with a finger in the vagina.

Treatment.—The important point in prevention of strictures is that following any operative procedure upon the rectum or anus the person should be kept under observation from ten days to a month and the finger passed in occasionally to note patency as well as any vicious healing that may occur. Cases of ulceration or any inflammatory lesion of the rectum should be examined occasionally by proctoscope, or at least by finger, to note if a stenosis is occurring. Most ulcerations of the rectum if diagnosed early could be checked in the destructive process and healed before they involve the muscular walls of the intestine.

Where distinct mechanical obstruction exists we may use gradual dilatation and forcible dilatation. For this purpose the finger and various forms of dilators such as the Wales bougie, Hagar's sound, Matthew's dilator, Beach's dilator, etc., answer to good purpose. In the early stages when the fibrous material is soft and elastic these may answer. A normal anal canal at its narrowest point measures about three centimeters in circumference. Above this the rectum dilates very rapidly and in its normal condition measures 5 to 15 centimeters. If the obstruction is below the reduplication of the peritoneum, forcible dilatation may be practised under anesthesia with no little success, but if the lesion is above this point great care should be exercised in the use of force. When employed it had best be done with a Matthews dilator protected with cotton or rubber tubing, and under vision through a proctoscope. It is best at all times that the dilatation be performed under the operative vision. Such a method is better than

the use of bags which cause pneumatic or hydraulic pressure, because of the impossibility of knowing how much force is being exercised. Rapid dilatation or divulsion of the stricture of the rectum is unjustified. When occuring rupture will always take place along the line of least resistance and this line is almost always toward the peritoneal cavity. Where the case seems suitable for dilatation this should be done at frequent intervals until the caliber of the gut and the strictured area is considerably widened.

Where the stricture cannot be dilated, or it is unwise to do so, various operations have been devised, the following representing the forms employed:

1. Linear incision with rapid dilatation.
2. Internal proctotomy.
3. External proctotomy.
4. Excision of rectum.
5. Colostomy.

The simplest of these is the linear incision, performed under a general anesthesia, in which the finger is used to guide a narrow bistoury with a straight blade which is passed above the stricture and indurated base. It is then turned toward the coccyx and is withdrawn, severing the cicatrix at that angle. Multiple incisions may be made in a circle; usually from three to six will suffice, after which a rapid dilatation with a Syms dilator bougie, and finally a thorough exploration with the finger to ascertain the complete condition of the scar tissue terminate the operation. A rectal tube covered with gauze is necessary to control the bleeding and makes the patient more comfortable. The tube is removed in forty-eight hours, the daily passage of soft bougies being practised until healing occurs.

The operations of proctotomy, excision of the rectum and colostomy, are found described in works on diseases of the rectum and anus and those on general surgery.

PIGMENTATION OF THE COLON AND RECTUM.

Chromatosis (von Recklinghausen's Disease) is in somewhat of a debate in medicine as to the cause, even as to the origin of the pigment. Opie believes that it is an entity. Adami thinks it both local and general, a hemachromatosis, which is an indication of destruction of red blood corpuscles extending over a long period of time with a subsequent heaping up of pigments, more particularly in the liver, and that the cells of that organ are incapable of dealing adequately with the iron containing a portion of the hemaglobin which thus re-

mains in a fixed state in the liver cells and other cells throughout the organism. The most recent work on this condition is by McFarland[5] who has pointed out that the pigmentation may be transitory and that it is in the connective tissue of the mucosa beneath the basement membrane of the gland epithelium and in the muscularis of the mucosa, in which cells it is found all the way from a very light brown to black, being in fine saccharine-like particles. In some cases the pigment is found in the solitary lymph-nodes and these can be seen beneath the muscularis. These pigments fall into two groups, exogenous and endogenous. Regarding the exogenous little need be said. They are usually either salts of silver or mercury, easily accounted for in the clinical history of the patient. Of the endogenous, however, a considerable study has been made; yet regarding them little is known. According to Pick they are caused from the aromatic albumin disintegration products of the contents of the large intestine (indol, skatol) under the influence of an oxidizing ferment resembling tyrosinase, produced by the connective tissue cells of the mucosa. This interpretation renders intelligible the limitation of the disease to the large intestine on the one hand and to the mucosa with its connective-tissue cells on the other. In such instances as I have seen at operation the pigmentation was always confined to the colon, this being the site of putrefactive processes provable at the time. According to Lynch, the condition is of bacterial origin and the extent of the disease is dependent upon the severity of the infection, the probable source of the infection is the intestinal tract, perhaps starting as an intestinal putrefaction, and this putrefaction lowers the vitality of the tissues and thereby the cells of the mucous membrane lose their protective properties, and consequently bacteria find ready access to the portal circulation. As a result of this the chromogenic function of the liver is interfered with and the liver becomes penetrated with pigment, with the result in addition that a certain amount of pigment is circulating in the blood. The conclusions of McFarland are:

"1. In true melanosis of the hind-gut, the pigment is not derived from blood pigment, but is a substance between true melanin and fat pigment.

"2. This pigment is probably formed by an enzyme which, manufactured by the intestinal contents, acts on the intercellular substance of the stroma of the intestinal mucosa.

"3. Pigment-containing cells are derived from plasma cells of the stroma of the intestinal mucosa.

"4. Intestinal pigment is primarily an accompaniment and not a cause of chronic obstipation.

"5. The clinical significance of intestinal pigmentation concerns the duration and intensity of obstipation, and, while a contributing influence in established obstipation, is not an initial etiologic factor."

The comment I would make on the above is that clinically none of the series seem to bear close analysis. In all instances of intestinal toxemia I make it a rule to examine the rectum with the proctoscope, and in over 5000 instances of definite cases pigmentation was met with but 21 times. In many of these it was observed as discreet deposits,

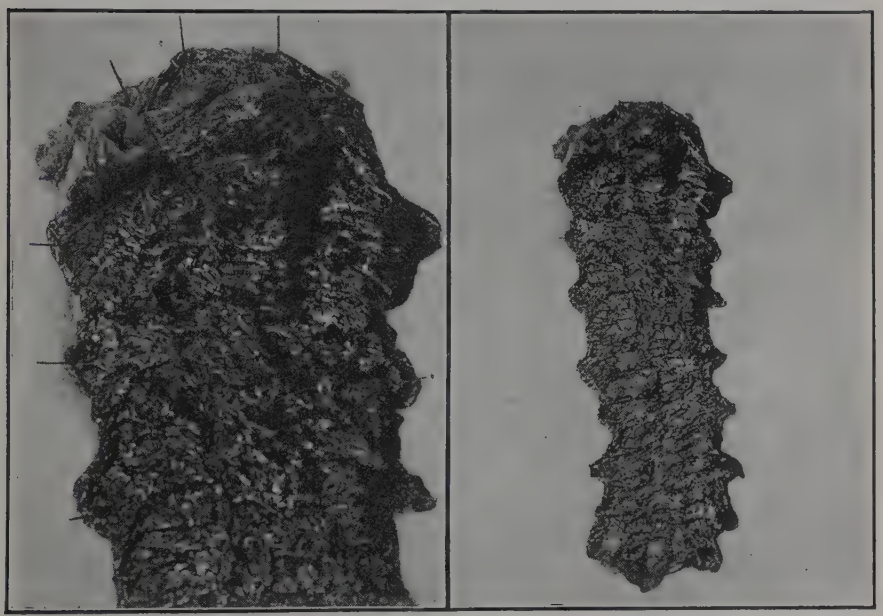


Fig. 154.—Deeply pigmented (black) colon removed from a case of marked intestinal toxemia, plus epilepsy. The mucous membrane of the rectum and the small intestine were not pigmented. The colon side of the ileocecal valve shown at the upper left side of specimen.

usually light in color, although three of them presented totally black mucous membranes. In the many more cases in which no intestinal toxemia was present and in which the rectum was examined proctoscopically, I have met with melanosis as often as in the toxemia cases, there being two in which perfectly black mucous membranes were found. What is interesting in this connection, although it may not have clinical significance, is that of the five cases of totally black mucous membrane four of them were epileptic, three from very early years and one acquired after the age of twenty-one in which there was

no intestinal stasis or putrefaction and no subjective or objective digestive distress or disorder.

Symptoms.—In my opinion there are no symptoms from the melanosis, and such symptoms as there may be from a condition in which melanosis is observed would not be due to the disturbance directly causing the melanosis. I am strongly inclined to feel that the condition presents no clinical aspect worthy of consideration, even though it be quite striking to meet with, and that in any clinical state in which this condition is found the treatment should be for the clinical condition present and not for the melanosis, because it invariably is found that after the clinical condition is cleared away, the melanosis still persists, and I have observed that the pigment persists for years after the clinical disturbance is corrected. To me it is simply a clinical curiosity without significance.

RUPTURES AND INJURIES OF THE RECTUM.

Nature has so well protected the anus and rectum in the bony framework of the pelvis that injuries of the organ from external causes are not common. However they are met with as contused, lacerated, incised and punctured wounds. Lacerations may follow the overstretching of the anal canal and manual dilatation of the muscles, the introduction of large foreign bodies into the rectum, expulsion of large fecal concretions, the passage of sharp foreign bodies in the stool, falling upon rough bodies like that of the root of a tree and the descent of a head in childbirth. The rectovaginal septum may be torn with complete rupture of the perineum in parturition and the head of the child may push the rectovaginal septum into the rectal cavity and the child be born through the anus with rupture of the septum, sometimes, however, without severe injury to the anus or perineum.

Symptoms.—There is no special or distinct symptom by which a diagnosis can be made. The history of an accident, the appearance of the part, the pain and the shock may give us a clue. Additional injury to one of the adjacent organs like the bladder or the peritoneum will give us symptoms peculiar to these organs. The hemorrhage may be slight and not show externally, although in all cases of injury the rectum should be entered, at least with a small tube, to see whether the cavity is filling with blood. The amount of shock is variable and is present in nearly every case. When the bladder has been injured there is pain in the region of the organ with disfunction of the bladder, and often the appearance of the urine will suggest whether that viscus has been injured or not by its containing blood or feces.

If the urethra be injured there will be a flow of urine from the rectum whenever the patient urinates.

Treatment.—The hemorrhage is usually controlled by pressure. If an inflammation upwards into the venous cavity as a phlebitis takes place, the usual result is almost inevitable. Ulceration, fistula, abscesses and incontinence may follow injury of the lower bowel. The most important feature in the treatment of wounds of the rectum is cleanliness and free drainage. In recent laceration of the rectum following delivery, or transverse rupture of the rectal wall by the passage of a large fecal mass, the parts should be united soon after the accident by suturing the rectal portion before uniting the vaginal surface. Punctured, lacerated or infected wounds, as well as those due to gunshot injury, the parts healing by suppuration, should be kept clean. Frequent douching should be done in all cases excepting in perforation of the bladder or the peritoneum, and in these cases the use of dry gauze is advocated. In cases of perforation of the peritoneum the abdomen should be opened at once and the parts thoroughly cleaned, the perforation closed, one end of the drainage tube placed in the site of the perforation and the other end brought out of the abdominal wound, and the patient placed in Fowler's position.

CONGENITAL ANOMALIES.

According to the statistics collected by Yeoman one baby of each 5000 born presents anorectal defect of development. The most common of these is imperforate rectum, in which the rectum may be absent or arrested in its descent at any point between the sacral promontory and a few lines above the anus.

The diagnosis of this condition is suggested by the absence of a normal canal, the condition being observed on examination. There may be a small dent representing an anal opening with an occlusion above it, therefore it is necessary to introduce the lubricated finger to see whether entrance into the rectum is possible or not. Another method is to inject water through the anal canal.

Various congenital formations have occurred in this connection, such as an imperforate rectum the anus being absent, an imperforate rectum with an anal canal well formed, and imperforate rectum with a vesical outlet, an imperforate rectum with a posterior urethral outlet, an imperforate rectum with a vaginal outlet, an imperforate rectum with spinal outlet, or a urogenital outlet in the rectum, or there may be simply membranous occlusion of the anus in which the anorectal membrane persists. The occlusion may be only partial in which instance it would run anteroposteriorly across the anal opening.

Treatment.—The treatment of these conditions is usually that known as proctoplastic, in which it is undertaken to make a rectum or anal canal. These plastic operations vary according to the anatomical condition present, and need not be entered into here. Colostomy has been suggested in some of the cases. As a rule, however, whatever form of treatment is used, the little ones do badly from the surgical procedure.

Malignant tumors (carcinoma), benign tumors, and venereal diseases of the anus and rectum are found in their respective chapters.

References.

[1] Whitehead: Brit. Med. Jour., 1882, i. 149.
[2] Murray: Jour. Amer. Med. Assn., Dec. 9, 1911, p. 1913-14.
[3] Ball: Brit. Med. Jour., Jan. 21, 1905, p. 113.
[4] Lynch: "Diseases of the Rectum and Colon," p. 213.
[5] McFarland: Jour. Amer. Med. Assn., Dec. 8, 1917, vol. lxix, No. 23, p. 1947.

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